

AN INTRODUCTION TO CHEST SURGERY

BY

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TO
THE STUDENTS OF THE LONDON HOSPITAL

PREFACE

WHEN I was a student I found two things wrong with most medical books—firstly they cost too much to buy and, secondly when bought proved difficult to read. These are grave faults in any kind of book, for the function of books is to communicate, and of introductory textbooks in particular to communicate agreeably. The passage of time has not made matters better in either of these respects, so I have taken the unusual course of writing this book in current English, hoping one day to see a student read it in the train, and the Oxford University Press has struggled to market it at a price most students and all doctors can afford to pay.

Had cost been no object, we should have liked to reproduce our X rays on a larger scale, but the printers have on the whole achieved clarity equal to that of much larger plates, and enabled us to make use of the rich variety of 128 films.

It is necessary in a book of this kind to be didactic, and I have set down the views personal experience leads me to believe are sound, and the methods and management I find successful. There are many alternatives, but this is not the place to discuss them, and both for this reason and for economy references are omitted.

Despite its concern with many of the commonest diseases, and the fact that in this field the greatest surgical advances of the past decade have been made, chest surgery is still regarded in many university hospitals as belonging to a special department—akin perhaps to neurosurgery—and the teaching of it is felt to be somehow less important than that of general surgery or medicine. The absurdity of this view should be made plain by a glance down the adjoining chapter headings. Yet to the best of my knowledge there is at present no other book dealing at student level with chest surgery, and it is in response to repeated requests of my own students that I have tried to do something to fill the gap.

I am grateful to my friends Mr Leslie Oliver and Dr W. Yell (both puritanical and penetrating semanticists), Dr Patrick Mounsey and Dr Donald Stride, for reading and criticizing sections of the text. In the wider prospect 'no man is an island' and surgical debts go back through all our predecessors to Hippocrates. I was fortunate in most of those who taught me chest surgery—at Brompton that great innovator and pioneer Tudor Edwards, allowed to die

unhonoured by a State quick to lavish titles on actors and Party hacks; later a taste of the superlative technical *panache* of Mr Holmes Sellors, still in the very van of progress; and finally an apprenticeship to my colleague Mr V. C Thompson, a number of whose methods I have adopted, and to whom I owe much.

Thoracic surgery is a taxing art. Students are often attracted by its drama, but daunted by its complexity. It belongs however to the main stream of surgery, not to one of its by-ways, and its practice calls only for the two qualities indispensable in any good surgeon—compassion and efficiency. Those who undertake it will be wise to follow Trudeau's precept. '*Soigner toujours; soulager souvent; guérir quelquefois*'

GEOFFREY FLAVELL

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July 1957

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Line drawings by Miss P. Archer

PART ONE

SURGERY OF THE LUNGS, PLEURA
AND THORACIC CONFINES

FIRST PRINCIPLES

CHEST surgery differs in principle from the surgery of other parts of the body only in that it is largely concerned with dynamic structures—the lungs and heart—upon which immediate survival depends. Otherwise it is directed, as is surgery in general, to the relief of obstruction, the extirpation of disease, and the preservation of healthy tissue. Many of the diseases with which it deals are among the very commonest afflicting the human body—cancer of the lung pulmonary tuberculosis, bronchiectasis lung abscesses, empyema, mitral stenosis, hiatus hernia, cancer of the gullet, to mention only a few. These are conditions with which every general practitioner is confronted daily. In the not very distant past some were treated medically others not treated at all. Today their treatment is surgical.

Surgery within the chest must first take account of (and preserve) the physiology of respiration. Providing sufficient oxygen is available on the one hand and haemoglobin is present to absorb it on the other, ability to breathe depends mainly on four mechanical conditions

- 1 An unobstructed airway
- 2 An elastic lung.
- 3 A partial vacuum between the pleural layers
- 4 A relatively rigid chest wall.

Let us consider these things in turn

1 The Airway

The airway is of course the trachea and bronchial tree itself. *Bronchial obstruction is just as much an acute surgical emergency as intestinal obstruction*, calling as urgently for immediate relief. The nearer to the trachea a blockage is, the more serious are the consequences the farther away the less severe. The bronchi lengthen and widen on inspiration, shorten and narrow on expiration. Thus air may pass a partial obstruction during intake of breath while the bronchus is wide, but be unable to escape again from the lung when it is narrow (see Fig. 1). The pulmonary distension which results is called obstructive emphysema (X rays 6, 62). This is a comparatively rare consequence of partial obstruction for it is not often that the block is of exactly the critical size needed to admit air during

inspiration and seal the bronchus on expiration. An almost invariable consequence, on the other hand, is the partial damming-up of mucous secretion beyond the obstruction. This pool of mucus soon becomes infected and converted into a pool of pus, which, as the blockage is not complete, is expectorated as purulent sputum.

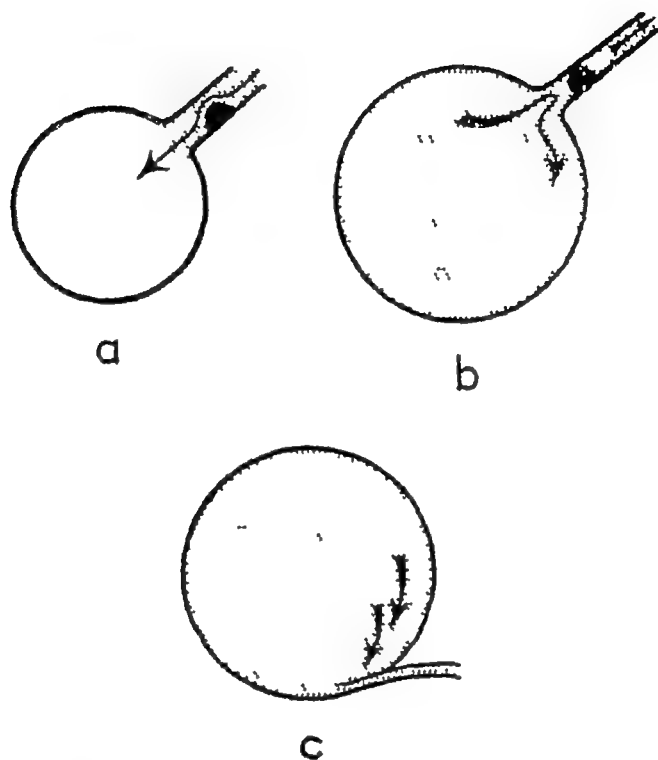


FIG 1

This is a diagram to illustrate how *partial* bronchial obstruction may produce obstructive emphysema or a ballooning cyst. In (a) air enters the lung or the cavity beyond the obstruction on inspiration because the bronchus dilates. In (b) the bronchus contracts during expiration so that obstruction becomes complete and air is trapped in the lung and distends it. By such a mechanism a positive pressure is built up.

In (c) a bronchiole enters an alveolar cyst obliquely, so that as the cyst distends it compresses the lumen of the bronchiole, turning it into a valve which again admits air only on inspiration. (See X-rays 36, 37, 39, 41, 42)

As soon as obstruction becomes *complete* such sputum *ceases*. Because drainage of the pus can no longer take place the patient is now likely to become febrile and ill, and if the infection is virulent a lung abscess or an empyema may follow. This sequence of events is seen in many disorders of the lung, and especially in bronchial carcinoma.

Other important consequences follow complete obstruction of a bronchus. The first is that air trapped in the part of the lung beyond the obstruction is rapidly absorbed, and the lobe or segment supplied by the bronchus becomes airless. This is the condition of pulmonary collapse or atelectasis constantly referred to through

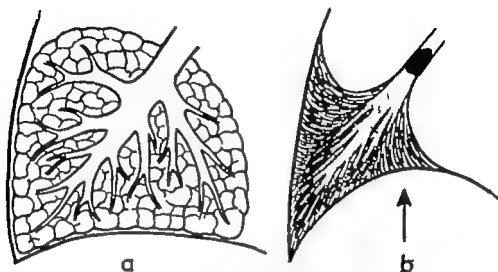


FIG 2

The effects of complete bronchial obstruction. A plug of mucus, or a growth, or a foreign body or extrinsic pressure, blocks the bronchus. At once the air in the segment beyond is absorbed and (a) contracts to (b). The bronchi are crowded together and filled with dammed-up mucus which distends them. The negative pressure in the pleural cavity increases, sucking the diaphragm and the mediastinum towards it and tending to draw the obstruction farther out into the bronchial tree.

out this book (see Fig. 2). The terms are synonymous, and must not be confused with passive *compression* of the lung caused by air or fluid in the pleural space. The pulmonary blood flow to the collapsed lobe continues, but the blood delivered to it can no longer be oxygenated. The bronchial mucosa continues to secrete mucus, but this is no longer able to escape and distends the bronchi in which it is trapped.

2 The Lung

The lung is an elastic sponge and is held out against the chest wall by the suction of the partial vacuum existing between the pleural layers. This negative pressure, which is naturally greater during inspiration and less on expiration, is usually between -7 to -15 cm of water. If the suction is broken by admission of air between the

pleural layers (as in artificial pneumothorax) the elastic lung retracts at once towards its hilum, and the mediastinum moves towards the opposite side of the chest. If a part of the lung collapses due to bronchial obstruction, the pressure in the closed pleural sac becomes more negative still, for the elastic drag of the lung upon it is no longer offset by the ebb and flow within it of air at atmospheric pressure. Thus the mediastinum is drawn *towards* the side of the collapse, and the diaphragm drawn upwards. In normal respiration there is a constant balance between the two forces of negative suction of the pleura and elastic retraction of the lung—rather like a man with outstretched arms exercising between two wall springs.

In old age, and in some diseases, the elastic quality of the lung is lost and breathing as a result becomes less efficient. If the lung is overstretched it also loses elasticity, so care is taken to avoid this whenever possible. The balance of forces is similarly upset by the presence of air or fluid between the pleural layers, and it is the constant preoccupation of the thoracic surgeon to get rid of them.

A further consequence of the lung's elasticity and of the tensions upon it is that if a hole is made in the mesh of the parenchyma it tends spontaneously to grow. Sometimes this tendency is greatly accelerated by the addition of positive pressure within the cyst or cavity so formed, because of partial obstruction of the air entry to it as in the fashion already described (X-rays 36, 41). The cavity distends until the pressure inside is sufficient to force air back the obstructed bronchus. If this is impossible, it continues to expand and may burst into the pleural cavity or progressively displace the mediastinum to the opposite side (X-ray 14). A functionless communicating with a bronchus, even if no pressure element is exerted upon it, may interfere with respiration in a manner of proportion to its size, for as well as preventing expansion of the surrounding tissue it entraps the tidal air in its dead space so that normal gas exchange is lost.

3. The Pleural Layers

The pleural layers, held closely together by the suction pressure between them, are moist, and glide one upon the other in the movements of chest and lung. If this freedom is lost by pleural adhesions and fusion, the efficiency of the mechanism is to some extent impaired. Effusion, bleeding, or the admission of air to the pleural space at once produces two effects—firstly, the vacuum (on which effective respiration largely depends) is lost, and the intrapleural pressure becomes much less negative.

positive, and secondly, as the lung is now only partially expanded its function is proportionately diminished. These facts are of constant practical importance in the treatment of thoracic surgical patients.

4 The Chest Wall

Lastly we come to the importance of a relatively rigid chest wall. Were the chest wall not rigid it would be impossible to maintain a negative suction within it and so to draw air into the lungs. Were it not rigid the impulse of coughing (which drives foreign substances from the bronchial tree by a brief blast of positive pressure), would instead merely puff out the flaccid chest. On the other hand if it were *absolutely* rigid respiratory excursion would be confined to the movements of the diaphragm. The moving ribs are the fingers which play the accordion of the lungs.

After trauma, and after some operations such as thoracoplasty parts of the chest wall are indeed left flaccid, either because ribs are comminuted or because they are removed. An area of this kind moves *paradoxically* i.e. it puffs *outwards* on expiration or on coughing, and is sucked *inwards* on inspiration as the negative pressure within the thorax is increased. If large, such an area is incompatible with life for not only is respiration seriously interfered with, but secretions can no longer be expelled from the bronchi. In addition the venous return to the heart depends to some extent on intra-thoracic suction and this too is impaired. A small defect can be tolerated for a limited period, but always renders the patient breathless and produces serious disturbance of function.

One should always think of the diaphragm as part of the chest wall—indeed, because it is the most mobile, the most important part. Although composed only of muscle and central tendon, it has a measure of rigidity imparted to it by the liver on the right, the stomach and spleen on the left, buffered against the positive pressure of the abdominal contents. A defect in the diaphragm, such as a hernial orifice and still more paralysis of one dome, is also a source of paradoxical movement, and consequently of breathlessness and impaired respiratory function. It therefore demands repair no less urgently than a defect in the thoracic cage.

In old age and in chronic disease of the lungs and pleura rib mobility becomes less and less as costal cartilages calcify, pleural layers thicken and muscles waste. Often the bony cage is completely rigid. When that time comes the machinery of respiration depends wholly upon the excursion of the diaphragm.

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SOME SURGICAL ANATOMY

THE anatomy of the thorax and the complex structures within it cannot be taught here, although details of it are later referred to and illustrated in the accounts of individual operations. In the chapter on 'Bronchoscopy' we will explore the branching of the bronchial tree itself. Anatomy is best learnt in the dissecting room and the operating theatre. Drawings help a good deal, written accounts very little. None the less certain practical aspects of thoracic anatomy must now be mentioned.

1. The Muscles

Although the pectoralis major arises from the upper part of the anterior thorax it seldom requires division at thoracotomy, for postero-lateral incisions pass below it, it is abducted from the line of antero-lateral incisions, and even in transverse section of the sternum and upper intercostal spaces it is merely split in the direction of its fibres. Only in the operation for pectus excavatum (see p 66) is part of its origin elevated from the lower costal cartilages.

On the other hand the whole back and flank of the thorax is ensheathed in the broad sweep of the latissimus dorsi which has to be cut across transversely in most thoracotomies. When this has been done the angle of the scapula is exposed with a triangle of fatty tissue below it bounded in front by the serratus anterior and behind by the lower fibres of the trapezius. The serratus radiates like a fan from the scapula so that its posterior fibres have to be cut across in postero-lateral thoracotomy, but the anterior part of the muscle is split (see Fig 11). To expose the sixth or seventh ribs both serratus anterior and a portion of the trapezius must be cut, but less and less of these muscles need be interfered with as ribs lower down are approached, for the triangle between them widens progressively. To approach higher ribs it is necessary also to cut some or all of the rhomboideus major attached to the vertebral border of the scapula; and to swing the latter forward, exposing the apex of the chest, the whole of rhomboideus and the inferior half of trapezius are divided, but there is no necessity to cut more than an inch or two of the posterior border of latissimus.

The external intercostal muscles slope downwards and forwards,

so that to detach them from the ribs the elevator is swept *forwards* along the *upper* border of the rib and *backwards* along the *lower* (see Fig. 12) True intercostal incisions (which cut across many perforating branches of the intercostal arteries) need never be made—if a rib is not to be removed the periosteum with its muscular attachments is simply stripped from the superior aspect of a rib and the incision made almost bloodlessly through the periosteal bed

The intercostal arteries themselves, arising from the aorta, are found posteriorly midway in the intercostal spaces between the necks and angles of the ribs, and here lie between parietal pleura deeply and external intercostal muscles superficially before they gain the subcostal grooves in which they run forward to anastomose with the internal mammary artery They are accompanied by the intercostal nerves and veins In aspirating the chest the subcostal position of these vessels must be remembered and avoided, the needle always being passed into an intercostal space in its lower part.

As a result of the diaphragmatic domes, parts of certain abdominal viscera, the kidneys with their suprarenals, much of the liver spleen and stomach lie within the thorax and may be injured by ill-directed attempts at aspiration or drainage, or by transthoracic missiles

2 The Ribs

In front ribs are counted, for purposes of localization, from the angle of Louis on the sternum which marks the second costal cartilage From behind they may be counted upwards from the twelfth, or from the seventh rib which lies just beneath the tip of the scapula. At operation the rib to be removed is more accurately identified by passing the fingers up under the scapula to feel the thick mass of the origin of serratus anterior from the second rib

The intercostal spaces are wider in front than behind, the upper ribs more horizontal than the lower which become progressively oblique as they descend, so that the anterior end of the fifth rib is at about the level of the posterior end of the eighth or ninth. One must therefore say which end of the rib is meant when using ribs to localize intrathoracic lesions In practice such references are nearly always to the posterior ends

Only the upper four costal cartilages articulate directly with the sternum in the line of the rib, below this point the cartilages become longer more curved, more indirect, and consequently more pliable The lower ribs are thus more mobile, the last two having no cartilages at all. Posteriorly all the ribs articulate with the vertebrae

by two facets, one on the head against the vertebral body, one on the neck with the transverse process.

The medial relations of *the first rib* from neck forward to costal cartilage are as follows. the cervico-thoracic sympathetic chain, the first dorsal contribution of the brachial plexus, the subclavian artery, the insertion of scalenus anterior into the scalene tubercle of the rib, the subclavian vein with the vagus posterior to it and the phrenic on its lateral aspect. The pleural dome also lies medial to the rib; and into the upper surface of the rib's anterior extremity is inserted the rhomboid ligament. These relationships are of great practical importance in thoracoplasty.

3. The Pleura

The pleura extends above the level of the first rib as a dome ensheathed in a continuation of the endothoracic fascia. This fascial dome—'Sibson's fascia'—is suspended by interdigitations of the deep fascia of the neck. It is in this position that the pleura is often accidentally punctured during operations on the base of the neck, especially on the stellate ganglion, the thyroid, or in dissection of tuberculous glands. Such punctures may result in haemopneumothorax.

Inferiorly the pleural sac just overlaps the twelfth rib behind, but extends down only to the level of the eighth in front. In this area the parietal pleura on the chest wall and on the diaphragm are in apposition, and if inflamed will adhere to obliterate the 'costo-phrenic angle'. The excursion of the lung within the pleural sac varies with each phase of respiration. The pleura covering the chest wall is easily stripped off the endothoracic fascia lying beneath it, but over the diaphragm it is densely adherent and difficult to remove.

4. The Lungs

The right lung is bigger than the left, for the bulk of the heart lies in the left chest. It has three lobes, upper, middle and lower in contrast to the left lung's two, upper and lower, the lingula segment of the left upper lobe—not present on the right—being the homologue of the middle lobe.

On the right, the front of the chest is related to the upper and middle lobes, on the left to the upper lobe alone (see Fig. 5). Posteriorly the upper lobes are related only to the apex of the chest, the great fissure between upper and lower lobes running obliquely downwards and forwards from the level of the fifth rib behind to the anterior part of the diaphragm in front. Most physical signs heard at the back of the chest emanate from a lower lobe, those in front

from an upper or from the middle lobe. On the right a horizontal fissure partially divides the upper from the middle lobe and runs forward from the hilum to the level of the fourth rib in front.

There can be no understanding of chest disease, chest X rays, and chest operations without a knowledge of the *bronchopulmonary seg*

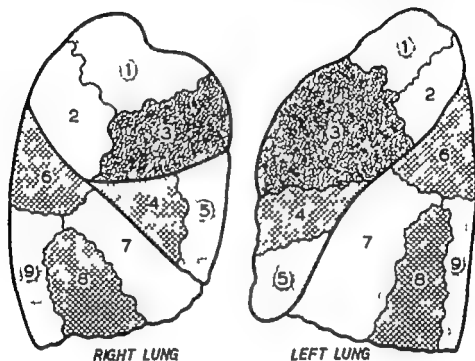


FIG 3

A lateral view of both lungs showing the arrangement of the lobes and bronchopulmonary segments. In the right lung the great oblique fissure divides upper and middle lobes from the lower; the horizontal fissure upper from middle. In the left lung no middle lobe or lesser fissure is present. In both right and left lungs (1) is the apical, (2) the posterior, and (3) the anterior segments of the *upper lobe*. On the right (4) is the lateral, (5) the medial segment of the *middle lobe*; in the left (4) is the superior division and (5) the inferior of the lingular segment of the *upper lobe*. In both (6) is the apical, (7) the anterior basal, (8) the lateral basal and (9) the posterior basal segments of the *lower lobes*.

ments (see Fig. 3) Their arrangement in either lung is similar save for the difference of lingula and middle lobe. Each has a separate bronchus (see Fig. 4) artery and vein, supplying a wedge of parenchyma distinct from its neighbours though not divided from them by fissures, as are the lobes themselves. Alveolar air leaks to some extent across these boundaries, which are marked by the branches of intersegmental veins. Each segment has a wider peripheral aspect

related to the pleura of the chest wall, the mediastinum or the diaphragm. Each can be removed by securing its individual bronchus and branch of the pulmonary artery, and peeling it from its neighbours, the segmental veins being ligated as they are brought into view. If this is accurately done, and adjacent segments are not diseased, bleeding and air leakage from the raw areas left behind are negligible.

The right upper lobe has three such segments, an *anterior* one related to the antero-lateral chest wall, a *posterior* one largely related to the upper part of the chest lying behind the scapula, and an *apical* segment occupying the pleural dome and thoracic outlet.

(An occasional anomaly affecting the right upper lobe but having nothing to do with segmental anatomy is the *azygos lobe* sometimes seen radiologically. This is formed by the azygos vein lying in a free 'mesentery' of pleura which deeply indents the upper lobe so that a part lies between the pleural sheet and the mediastinum. It is of no pathological importance.)

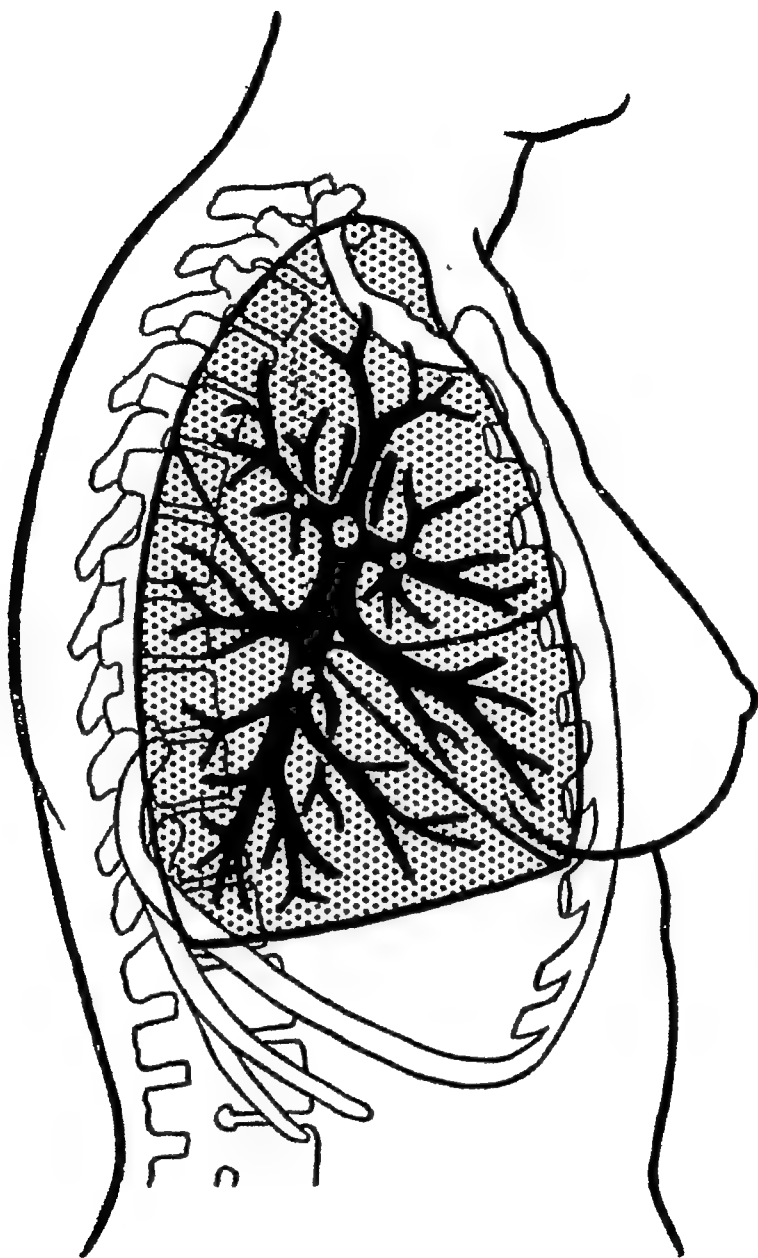
The middle lobe has two segments, *medial* and *lateral*.

The right lower lobe has first, an *apical* segment, chiefly related to the posterior chest wall below the posterior segment of the upper lobe, a small *cardiac* or *medial basal* segment lying on the mediastinum behind the convexity of the right atrium as seen on a postero-anterior X ray, and three larger basal segments, *anterior*, *lateral* and *posterior*, the anterior being related to the aspect of the middle lobe abutting on the oblique fissure, the lateral to the axillary chest wall in its lower part, the posterior to the back below the area of the apical segment of the lower lobe, and all three inferiorly to the diaphragm (X rays 25, 26).

The left upper lobe differs from the right in two respects. It possesses in the *lingula* an additional segment, the first of its branches, lying antero-medially in front of the pericardium and reaching almost down to the diaphragm, and its apical and posterior segments share at first a common bronchus. There are thus, in addition to the lingula (which has a superior and an inferior division), *anterior* and *apico-posterior* segments of which the anatomical relationships are similar to the corresponding segments of the right upper lobe.

The left lower lobe is in all respects the same as the right, except that no cardiac segment is present—only *apical*, and *anterior lateral* and *posterior basal* segments arranged similarly to those on the right (see Figs. 3, 4 and 5).

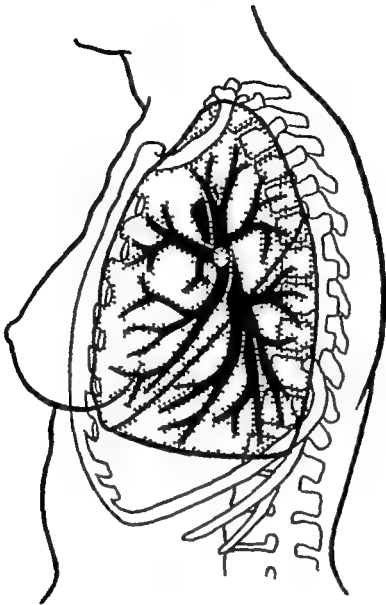
The relationship of the hilar structures entering the lung is dealt with in the description of 'Pulmonary Resections' (see Figs. 13, 14, 17, 18, 19 and 20).



RIGHT LUNG

FIG 5

Lateral views of right and left lungs to show the relationship of the lobes and segmental bronchi to the chest wall. Note how the *lingular segment* of the left upper lobe has similar relations to those of the *middle lobe* in the right



LEFT LUNG

Distinguish the apical, anterior and posterior segments of each *upper lobe* the apical, and three basal segments of each *lower lobe*—anterior lateral and posterior. On the right side the additional cardiac, or medial basal, segmental bronchus is visible.

HOW TO LOOK AT A CHEST X-RAY

EXAMINATION of the chest is incomplete without radiography. Inspection and physical signs may tell much—but seldom all—about disease within, but often they tell nothing, and fatal lesions are ignored through failure to obtain an X-ray. No doctor sends away a patient suspected of having a fracture without radiological proof; no doctor ought to send away a patient with symptoms of chest disease without it. In the one a limb may be at stake, in the other a life.

It is even worse for an X-ray to be taken and for a serious abnormality in it to be missed. Success in reading chest X-rays depends not upon *expertise* but upon conscientious observation. Radiological subtleties—‘Doctor So-and-so’s Lines’—‘perhaps a *little* narrowing of those spaces’—are seldom of importance. The things that matter are usually clearly visible to anyone who looks at the film systematically and carefully. What excuse is there for missing the fact that the whole lower lobe of the left lung is collapsed? For calling a carcinoma ‘unresolved pneumonia’? For mistaking a breaking-down growth for a pyogenic abscess? Yet these things happen—and sometimes in the most exalted circles—every day.

To omit a lateral radiographic view, while inspecting a postero-anterior one, is the equivalent of examining a patient only from in front. Not only is it then impossible to say whether a tumour is anterior or posterior in the thorax, or in which pulmonary lobe it is, but also lesions obscured by the curvature of the diaphragm or by the heart shadow may pass unnoticed.

A chest X-ray must satisfy certain technical requirements. They are as follows:

(1) It must be well centred—i.e. in a P-A view the clavicular heads should be equidistant from the vertebral spines. Failure in this respect results in apparent cardiac displacement to one or other side, alteration in the prominence of hilar shadows, and, especially in infants, artifactual mediastinal ‘masses’. Similarly postero-anterior views should always be taken at 6 feet, so that the apparent heart size is not altered by differing focal lengths. At 6 feet the normal heart is not more than half the chest width.

(2) Penetration must be adequate, and constant for all subsequent

films. The most satisfactory penetration for surgical work is that which just permits delineation of the vertebral bodies through the heart shadow. Unless successive radiographs are comparable they serve only to deceive. Lesions appear to melt away in views of greater penetration, grow bigger when penetration is less.

(3) Focus must be sharp

(4) Bandages, dressings, pyjama jackets, braids of hair, necklaces, crucifixes, amulets, braces and all other extraneous foreign bodies must be removed. The shoulders are rotated outwards so that the scapulae do not overlie the lung fields.

Should chest X rays fall short in any of these particulars no conclusions should be drawn from them—they should be repeated.

The first, and most important, step in the interpretation of chest radiographs calls for one thing—ability to recognize normal appearances. This can be acquired by anyone (and certainly any medical student) who makes a practice of looking, not at X ray reports, but at X rays. It is essential that study of chest films is *always systematic*.

(1) Having satisfied yourself that the picture is well centred, well penetrated, and sharply focused, look first at *the bones and soft tissues outside the thorax*. Is a fracture, or bone erosion due to a secondary deposit, visible in the scapulae or either clavicle? Do the dark streaks of surgical emphysema outline the tissue planes?

(2) Next comes the *general shape of the thorax* itself. Is scoliosis present, causing rotation and apparent displacement of the heart? Is the chest barrel-shaped, with widened subcostal angle as in chronic emphysema? Is it symmetrical? Perhaps one side is notably smaller than the other—the ribs more sloping, the intercostal spaces narrower. This at once suggests the presence of chronic disease in the underlying lung.

(3) Examination of the whole of *the bony cage* must never be omitted, each rib being compared with its opposite number from top to bottom. Ribs are counted by their *back ends*, beginning with the first. Accuracy is essential for upon it the localization of intrathoracic lesions depends. Are congenital anomalies of the ribs present, or is a cervical rib (which may be the cause of symptoms) visible? Congenital deformities here may accompany congenital abnormalities elsewhere. Are the ribs horizontal and widely spaced as in emphysema, or oblique and narrowed as when chronic disease underlies them? Is a fracture to be seen—and is it a pathological one (X ray 59) or traumatic or due to cough? Have ribs been resected in the past? Are their under-edges notched by anastomotic vessels as in coarctation? Are any ribs eroded by growth, or by a metastasis

or simply by pressure? Not long ago I saw a patient with an opaque rounded shadow abutting on his lower ribs (X-ray 65). A diagnosis of encysted pleural effusion had been made by the radiologist, and accepted by the physician. Repeated attempts had been made to aspirate it. Examination of the bony cage in the X-ray showed parts of these ribs to be missing—they were completely eroded, and the opacity was therefore a carcinoma. The diagnosis depended not on skill but on the most superficial observation. Lateral films give a better view of the vertebral bodies, and of the outline of the sternum.

(4) *The diaphragmatic domes* are inspected. The right is normally a little higher than the left. Beneath the left a bubble of gas is usually visible in the stomach. General elevation of one dome may be due to phrenic paralysis and calls for confirmation by screening. Elevation of only part of one dome may be due to inflammatory adhesions, to subphrenic abscess, or to eventration. The cardiophrenic angle, made by the convex right borders of the heart shadow and the right dome, is an acute angle. Its obliteration may signify pulmonary collapse (X-rays 46, 58) The costophrenic angles, where diaphragm meets chest wall, are also acute. Their flattening means that the adjacent pleural layers have become adherent, i.e. that the patient has at some time had pleurisy (X-ray 21) But should they be obscured by an opacity curving up from the edge of the diaphragm towards the axilla as a concave meniscus, the likelihood is that a pleural effusion is present. On lateral films the two diaphragmatic domes are superimposed on one another, but their anterior and posterior margins can be seen, and any irregularities of their surface localized.

(5) *The mediastinum* is of great importance, for upon its displacement diagnosis of intrathoracic abnormalities often depends. Its central position is altered for all practical purposes only by one of three causes:

- (a) The X-ray is not straight.
- (b) The patient has scoliosis
- (c) Disease is present in the lung or the pleura.

Providing the first two of these have already been eliminated shift must either be due to the mediastinum being *pushed away* by air or by fluid in the pleural cavity (X-rays 1, 13, 14), or *pulled towards* an area of pulmonary collapse (X-rays 2, 3) or fibrosis (X-ray 29). Of these *collapse* is incomparably the most common and the most important for it is *always* of serious, and sometimes of fatal, significance. It is more often misdiagnosed, or missed altogether, than any

other lesion of the lung. It masquerades as postoperative pneumonia interlobar effusion and pleural thickening. Such errors will not be made if the position of the mediastinum relative to an opacity is observed. Compared to collapse fibrosis is a rare cause of mediastinal displacement. It occurs in patients with chronic pulmonary tuberculosis and long-standing inflammatory lesions such as gross bronchiectasis or suppurative pneumonitis (X ray 76). The mediastinum is pushed to the opposite side by pleural effusions, empyema, haemothorax, or by a pneumothorax (X rays 1 13 14). Rarely it may also be displaced by a large solid tumour.

As a first step in diagnosis, therefore, the normal configuration of mediastinal structures must be known. Beginning at the top one sees the translucent (because it contains air) outline of the trachea passing down between the equidistant clavicular heads. The right border of the superior mediastinum is straight almost until the hilum of the lung is reached. A bulge above this point suggests a tumour—the most common being metastatic enlargement of lymphatic glands although many other conditions may give rise to one. At the level of the hilum of the right lung is the gentle convexity of the ascending aorta. Below this, and extending to the cardiophrenic angle, is the much more prominent convexity made by the right atrial wall. Normally about one-third of the heart lies to the right of the midline two-thirds to the left. *Disappearance of the right atrial shadow with emergence of the straight line of the vertebral bodies signifies displacement of the heart to the left* (X ray 2). The commonest cause of this, by far, is collapse of the lower lobe of the left lung. Upon recognition of this simple fact a patient's life may often hang. Of all crucial pulmonary calamities this is the one most often missed.

The left border of the superior mediastinum is also straight until the aortic knuckle is encountered. Its prominence varies with cardiac and vascular disease and sometimes it lies instead upon the right as a congenital anomaly. Below this, and just above the hilum is the lesser bulge of the pulmonary conus with a further slight convexity below made by the atrial appendage. Finally comes the wider sweep of the border of the left ventricle.

The lateral X ray allows any mediastinal abnormality to be accurately localized and upon this the diagnosis of intrinsic tumours depends. In the superior mediastinum the clear tube of the trachea is again seen running downwards and slightly backwards. Anterior to this, and just behind the sternum, is a notably translucent area where the mediastinum consists virtually of two pleural layers between which lies the thymus. It is through this area that herniation

of one lung may occur into the opposite pleural cavity. Behind this point and above the hilum of the lung curves the arch of the aorta. The position of the hilum is clearly marked by the clear, round, central translucency where the main bronchus, or the bronchus to an upper lobe, is seen cut across. In the lower part of the anterior mediastinum lies the pear-shaped shadow of the heart

(6) *The lung fields* are studied and compared Just as is the chest as a whole, so must the lung itself be surveyed systematically. The shadows at the hilum, which fan out towards the periphery of the lung, are due to the pulmonary artery and vein They are more prominent on the right than on the left, where they are overshadowed to some extent by the heart itself. It is important to learn, by experience, their normal appearance, for small growths may be superimposed on them They are both more conspicuous, and actually larger, when the surrounding lung is emphysematous. Recognition of emphysema in the lung to be left behind may be of prime importance when pulmonary resection is contemplated. The lung visible in each intercostal space is compared with that on the opposite side, *space by space, from top to bottom* If this is always, and conscientiously, carried out differences and abnormalities cannot be overlooked An obvious lesion in one lung must not divert attention from the opposite side, for perhaps there will be found a secondary deposit, a spill-over abscess, evidence of tuberculosis, or some other vital fact which alters treatment or confirms a diagnosis On lateral films the interlobar fissures may be visible

(7) Finally *the abnormality itself*, if one is present, is considered. Does it appear to be in the lung, in the pleura, or related to one of the surrounding structures? This is not always easy to decide, and may depend on other investigations, but the lateral X-ray is often conclusive. If mediastinal displacement has been detected, one already knows whether an opacity is due to fluid or to collapse, and this is a long step towards diagnosis. The shadows caused by lobar collapse are characteristic (see Fig 6 and X-rays 2, 3, 4, 5, 46, 58). Even in the absence of mediastinal shift, an opacity which is adjacent to the mediastinum, and which has a concave edge bordering the lung, strongly suggests collapse.

A shadow in the lung should be described in much the same way as is a lump in the breast Firstly, where, precisely, is it? In which segment of which lobe? Or is it trespassing from one segment, or one lobe, to another? The position of the interlobar fissures, and the outlines of segmental anatomy, must always be present in the mind What is the edge of the shadow like—clear-cut and regular as is that

of an innocent neoplasm, or vague and ill-defined like that of an invasive one? What of the consistency—is it dense or translucent? Is calcium present in it? Does it appear homogeneous, or is it cavitated? The most dramatic and easily recognizable shadow in chest radiography is the sharp horizontal line of a fluid level (X rays 22, 23 24 33, 34 35 49 52, 53) Such a level proves the presence, not only of fluid, but also of air. Air can enter in one of three ways

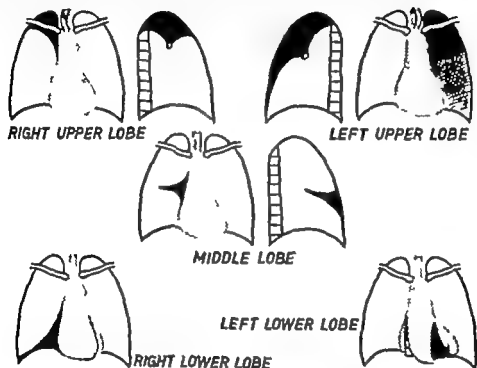


FIG 6

The characteristic radiological opacities produced by lobar collapse. Note how the opacity in left upper lobe collapse affects almost the whole lung field in posterior-anterior views, for no middle lobe is present. Note also how the shadow of left lower lobe collapse lies completely behind the cardiac opacity

—from a bronchus, from the bowel, or by being let in through the chest wall. If fluid communicates with a bronchus it must be present in the sputum. If it communicates with a bronchus fluid is likely to be infected

If these methods, and these principles are kept in mind it is not difficult to form an intelligent opinion about a chest X ray. In the telling they sound lengthy and laborious with practice systematic orderly observation becomes easy. It is indispensable. Without it fatal lesions will sooner or later be missed with it very few pass undetected.

Special techniques

(1) *Radiological screening* is essential for further investigation of most patients. By demonstrating mobility with respiration it helps to differentiate tumours in the lung from those attached to the chest wall or mediastinum, and it detects pulsation in them. Diaphragmatic movements are tested (by asking the patient to sniff) to see if one or other dome is paralysed, the affected side moving *paradoxically* (i.e. up instead of down). The swallowing of barium also yields useful information about the relationship of tumours to the oesophagus, lesions of the oesophagus itself, and cardiac enlargement.

(2) *Tomography* provides serial views focused at intervals (usually of 1 cm.) from front to back, or from side to side, throughout the substance of a lung. It enables the character of a tumour or of a diseased area of the lung to be studied in detail, as well as fixing its relationship to neighbouring structures with great accuracy (X-ray 55).

(3) *Kymography* is a radiological technique which records the degree of pulsation in the heart, great vessels, or in tumours.

(4) *Bronchography*, the instillation of radio-opaque oil into the bronchial tree, is of wide application in the study of pulmonary disease and is fully described on p. 118.

BRONCHOSCOPY

THE bronchoscope is the most important single instrument used in thoracic surgery essential for diagnosis, indispensable in treatment. There is virtually no pulmonary disease about which it will not yield valuable information, and the thoracic surgeon approaches his patients bronchoscope in hand, for it is to him as the cystoscope is to the urologist or the proctoscope to the rectal surgeon. The interpretation of endobronchial appearances calls for much specialized experience and skill but the use of the bronchoscope to relieve post operative collapse of the lung, and the accumulation of bronchial secretions, should be within the range of every registrar and every practising general surgeon without exception and the necessary technique is safe—much safer than oesophagoscopy or even sigmoidoscopy—and is easy to learn. To allow a patient who has had a successful abdominal operation to collapse part of his lung afterwards, and perhaps die from it, or at best develop a lung abscess or bronchiectasis, and to do nothing about it or simply shrug it off as hypostatic pneumonia is a crime when the condition can so easily be cured, or better still prevented, by prompt bronchoscopic aspiration.

There is no age limit for bronchoscopy and it can be carried out on anyone. It is applicable to the suckling or to the senile to the well or to the very ill, and can be performed in the operating theatre or in bed in the ward, or with the patient sitting upright in a chair. A patient is never too ill to be bronchoscope. If bronchoscopy is likely to help him. Except in infancy only local anaesthesia is usually required. There are many conditions in which bronchoscopy is imperative and frequently life saving its only contra indication is the recent ingestion of a meal, and even this is not absolute.

Should a man over 40 have a haemoptysis or a fixed rhonchus be audible in his chest, or should he suffer from a lung abscess or an empyema, or from 'unresolved pneumonia' or have a persistent abnormal opacity of any kind in his chest X ray the omission of bronchoscopy amounts to culpable negligence, for only by it can a cancer be found in time. No investigation of lung disease is complete without a bronchoscopy any more than it is complete without a postero-anterior and lateral X ray of the chest. It provides the only

direct access to the bronchial tree and so enables one to study impairment of its normal mobility or of the aeration of any segment, to see inflammation or ulceration of the mucosa; to discover a source of bleeding or of pus; to remove secretions from any part for bacteriological or histological study; to localize pressure on the bronchial wall from without, and to inspect strictures or frank growths and to identify them positively by biopsy.

Technique

The Chevalier Jackson bronchoscope with distal lighting is the best type of instrument because it is simplest and gives the maximum field of vision in relation to its diameter. There are several sizes, ranging from suckling, infant and adolescent, to small and large adult, but the larger the instrument the easier is the bronchoscopy and the largest size suitable to the patient should always be employed. A powerful and reliable source of suction, and a selection of wide-bored aspirating tubes are essential to safety, and bronchoscopy ought never to be attempted in their absence. For the relief of obstruction due to accumulated secretions no more is required, but for general diagnostic use there must also be at least one right-angled telescope, a variety of biopsy and grasping forceps, a trap for fluid samples, swab holders and preferably (but not necessarily) an adjustable bronchoscopic head rest.

The patient should have nothing to eat or drink during the four hours preceding bronchoscopy unless it is an emergency one. Where small children are concerned it should be remembered that food often remains undigested in the stomach for much longer periods if they are frightened and ill, to be inconveniently regurgitated when least expected. Under general or local anaesthesia such vomit may suddenly be aspirated into the trachea and cause death by drowning or by the impaction of solid fragments and such a contingency itself calls for the prompt passage of the bronchoscope and for vigorous suction to clear the airways, solids sometimes having to be extracted with forceps. Accidents of this kind are already sufficiently common, so they must not be allowed to occur during bronchoscopy, when instrumentation of the pharynx may promote gagging and retching, and an anaesthetized glottis render aspiration fatally easy. It is for this reason that the stomach should be empty, anaesthesia efficient, instrumentation gentle and practised, suction powerful and suction tubes wide and quickly interchangeable, and some means available for rapidly lowering the patient's head if necessary. If these conditions are observed, the dangers against which they guard will not occur.

The choice between local and general anaesthesia will depend upon the age and temperament of the patient, but in the very great majority local is to be preferred as it is completely efficient if properly administered permits 8 to 10 bronchoscopies to be performed in an hour without the services of an anaesthetist, and does not involve patients remaining in hospital. A bronchoscopy session is an essential part of any thoracic surgical out patients clinic.

Premedication is not necessary, but in patients or unusually nervous subjects are given Omnopon gr $\frac{1}{2}$ and hyoscine gr $\frac{1}{100}$ an hour beforehand. The anaesthetic itself is begun by the patient sucking a 50-mg. Nupercaine tablet on the back of the tongue twenty minutes prior to operation. On arrival in the theatre the patient sits up and extends his neck. After the skin over it has been sterilized the cricothyroid membrane is felt, a fine intramuscular needle passed through it into the tracheal lumen, and exactly 1 cc. of 10 per cent. cocaine hydrochloride rapidly injected with the patient leaning to the side of principal interest. The needle is withdrawn, and the cocaine is partly coughed up on to the vocal cords and partly trickles down over the bronchial mucosa. A pledget of ribbon gauze, grasped between the teeth of a Krauss forceps is then dipped in 10 per cent. cocaine, and after the surplus has been carefully squeezed out, is passed over the back of the tongue, which is gently drawn forwards with a swab down into the pyriform fossa where it is held for a moment or two to anaesthetize the glossopharyngeal nerve endings. One such application only is used on either side and never repeated. All the cocaine used is carefully measured from a graduated glass, and never exceeds 2 cc. Under no circumstances should the back of the throat be sprayed with it, or indeed with anything else, for accurate measurement at once becomes impossible. In the past decade I have employed this technique about eight thousand times without the slightest mishap or trouble and have myself never encountered an example of cocaine sensitivity. On the other hand I have seen several patients killed by sprays and solutions carelessly slopped about. Cocaine shares with Pentothal the position of medicine's most abused drug both are in common use both are too easy to give both are responsible for many deaths. Cocaine sensitivity must be exceedingly rare cocaine poisoning by overdose appallingly frequent.

Some bronchoscopists prefer to use one or other of the many proprietary cocaine derivatives such as 2 per cent. Butyn or 4 per cent. Xylocaine on the grounds that their toxicity is less. Though this is true intelligent witnesses who have experienced these alternative drugs strongly favour cocaine as an anaesthetic and it should be

lateral and *posterior* are arranged in similar fashion to those on the right (see Fig. 8).

When a complete examination has thus been made and all the primary divisions identified and inspected to the limit of vision, the bronchoscope is withdrawn to the trachea, observations being confirmed on the way, and it is finally removed altogether in an upwards and forwards sweep. As his pharynx and glottis remain anaesthetized for some time, the patient must not eat or drink for an hour and a half after the bronchoscopy, when the effects of the cocaine will have worn off and he can sip water without spluttering.

Therapeutic uses of bronchoscopy

1. *The relief of postoperative collapse*

After all operations, but especially those upon the upper abdomen and the chest, there is a tendency for part of the lung to collapse. Following laparotomy the affected part is usually basal; but in thoracic work it is generally the lobe or segments left behind after a resection, or the lower lobe after a relaxant operation such as thoracoplasty. The explanation is quite simple. As it hurts the patient to cough he does not do so, secretions accumulate in the lower lobe bronchi, the air beyond is absorbed, and collapse follows. There is then no way to expel the mucus, and even if the patient can be persuaded to cough his efforts are likely to be ineffective. Two other factors are often added: if the patient lies unconscious or is befuddled for a long time after returning from the theatre, or is too heavily sedated, secretions accumulate and will not be coughed up; and if he is allowed to become dehydrated, as is very often the case, the mucus becomes sticky and tenacious and exceedingly difficult to shift. It is common to find the actual mucoid plug in the blocked bronchus so tough and dry that the strongest suction is unavailing and it has to be extracted with biopsy or grasping forceps. Such plugs are quite rubbery and gelatinous, and can be rolled between the fingers; so that their expulsion by coughing or by postural drainage is inconceivable. Even if the patient is fully conscious but lies inert, perhaps frightened to move much or because he finds it painful to do so or, unforgivably, because someone has told him to, the same thing will happen. All this can be prevented: (1) if patients are taught, and helped, to cough regularly and as soon as possible, after an operation; (2) if anaesthesia and sedation are not excessive and the return to consciousness prompt; (3) if *enough* sedation is given to dull pain but not wits; and (4) if early movement both in and out of bed are encouraged. I remember during the last war when innumerable her-

niotomies were performed on military recruits, the incidence of pulmonary complications variously diagnosed (but all in fact one degree or another of pulmonary collapse) was shockingly high, and many learned articles were written advocating local anaesthesia instead of general, or spinal instead of local, and later many more indicating the trouble was just as bad with any of them. The real nigger in the wood pile was that the patients were kept lying flat on their backs for weeks on end instead of getting out of bed the next day as they should have done.

After resection of lung, especially lobectomies, these difficulties are intensified by major trauma to the chest wall itself by the accumulation of air or fluid in the pleural cavity and by oedema in the bronchial stump helping to narrow the lumen. This is worst about the third or fourth post-operative day, and this is the time such collapses are most common. The less co-operative or intelligent the patient or the poorer his physical condition the more likely are they to occur and children and rather silly teen age girls have the highest incidence of atelectasis. A normal cough displaces about three pints of air in a second, and this is the tussive blast that expels secretions, but once the lung behind is airless not only is there no force left to drive secretions out but a negative drag is established in the collapsed lobe or segment which tends to drag them farther and farther out in the bronchial tree and make their removal hourly more difficult (see Fig. 2)

As soon as collapse has occurred, therefore or someone has diagnosed postoperative basal consolidation or hypostatic pneumonia, bronchoscopy is not only indicated but becomes a matter of urgency. The same applies to patients who have not yet got collapse but who for one reason or another cannot cough up secretions, which are obviously accumulating, rattling distressfully up and down in the trachea and setting up a vicious circle of their own. This is seen where a degree of heart failure follows surgery, or in myasthenia gravis, or in poliomyelitis and the relief brought by bronchoscopic aspiration is not only dramatic but often by breaking the circle saves the day. *Patients are never made worse by bronchoscopy.* The condition of the patient should not be a deterrent but a spur, and an old or ill or feeble person is much more likely to die of slow drowning by being left alone or of pulmonary collapse than of the transient discomfort of bronchoscopy. In such cases the instrument can conveniently and swiftly be passed while the patient is in bed in the ward, providing only that good suction is available there. As soon as the pus and mucus have been sucked out, or any solid plug extracted

with forceps, the patient coughs as vigorously as he can so that peripheral secretions are driven in towards the sucker; and a finer sucker-end, soft-tipped, can be passed on into smaller bronchi to ensure complete clearance. The result is instant relief and the prompt re-expansion of the collapsed lung. In addition the patient is saved from possible lung abscess, or empyema, or the later development of bronchiectasis. Such treatment ought not to be confined to the realms of thoracic surgery: it is an essential part of all surgical after-treatment; and could be employed with benefit in many medical wards.

2. In the treatment of suppuration

Bronchoscopy is always indicated in the early stages, and sometimes in the later, of an aspiration lung abscess to remove any slough that may be interfering with free drainage and to establish that no organic obstruction such as a growth or a foreign body is present. This is also occasionally necessary in bronchiectasis, but repeated bronchoscopy merely to aspirate pus has little to recommend it, for the end is better achieved by postural drainage and antibiotics, or by surgery. The practice of insufflating antibiotic powders into an infected portion of the lung by means of a bronchoscope is ineffectual, unnecessary and dangerous, for the powder itself may act as a bronchial embolus.

Should an infected dermoid, or hydatid, or bronchogenic cyst rupture into the bronchial tree, or an empyema or even a subphrenic abscess establish a fistula and flood into the lung, bronchoscopy may be life-saving; as it similarly is in saving a child from drowning who has inhaled its vomit.

3. In the treatment of tuberculosis

Bronchoscopy is chiefly necessary in tuberculous patients for diagnostic purposes and preoperative assessment. Tuberculous strictures of the bronchus, for example, are an indication for resection, and are seldom satisfactorily treated by bronchoscopic means. When a child's bronchus is compressed causing collapse or, less often, obstructive emphysema, it is commonly by enlarged tuberculous glands outside, which press upon the relatively soft bronchial wall which in early life contains only pliable cartilage or none at all. Attempts to dilate such a stricture are futile for as soon as the dilators are withdrawn the wall falls in again. Intrinsic strictures caused by endobronchial tuberculosis in older patients are always associated with permanent pulmonary damage beyond which calls for removal; but



1

X-ray 1. A substantial left pleural effusion. Note the way in which the fluid rises in the left axilla and the displacement of the heart and mediastinum to the opposite side.



2

X-ray 2. Collapsed left lower lobe in girl with bronchiectasis. Note absence of ariol convexity right of the vertebral column. The darker triangle of collapsed lobe seen through the heart shadow is usually invisible, diagnosis depending on cardiac displacement alone. (See also X-ray 27)



3

X-ray 3. Typical postero-anterior appearance of collapse of the upper lobe of the left lung. Note how the area of opacity extends almost to the diaphragm.



4

X-ray 4. Lateral view of a collapsed left upper lobe. The area of opacity lies in the front of the chest, limited by the interlobar fissure which here extends obliquely from the apex, through the hilum, down to the diaphragm anteriorly. The apex of the lower lobe therefore is situated in the top of the pleural cavity.

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as a purely palliative measure, when surgery is for some other reason contra indicated, dilatation may be justified, though it should never be attempted if active disease of the mucosa is still present. Rarely tuberculous glands ulcerate through the bronchial wall to spill their caseous contents or to present, when calcified, as broncholiths and be delivered bronchoscopically.

Cauterization of tuberculous ulcers gives poor results for the ulcers indicate widespread infection of the submucosal layers, and cautery applications are more likely to be followed by stricture than by healing.

4 *In the treatment of new growths*

Before resection was widely practised bronchial adenomas were treated by repeated bronchoscopic fulguration to clear the lumen and allow re-expansion of the lung beyond. This sometimes made life tolerable for many years, but there is no place for it in modern treatment if resection is practicable. It is well calculated to promote malignant change and in any case permanent damage to the distal lung is almost inevitable. Similarly the insertion of radon seeds into the intrabronchial portion of a carcinoma has nothing to recommend it. None the less, cases of acute obstruction due to neoplasm occasionally arise which can only be relieved by bronchoscopy. An Indian woman was in a state of acute respiratory distress and rapidly becoming worse. On arrival I found her almost *in articulo mortis*, deeply cyanosed and taking periodic crowing inspirations. A bronchoscope was passed without any form of anaesthesia, and in the trachea a large pedunculated mass was encountered which flapped back on inspiration and impacted on expiration. It was seized with biopsy forceps and enough was torn from it to re-establish the air way, giving instant relief. It proved to be an ulcerating protrusion of lymphadenoma, and in fact the patient is alive six years later.

5 *In anaesthesia*

Bronchoscopy plays an essential part in the administration of anaesthetics for chest surgery. For many resections either of lobes or whole lungs, it is desirable to block off the portion of the bronchial tree to be amputated by the accurate placing of an endobronchial balloon or blocker. This is sited under direct vision at bronchoscopy and subsequently inflated so that it is firmly gripped and retained in position after the bronchoscope is withdrawn. A suction tube passes through the blocker itself so that pus from the distal lung can be aspirated. When the operation is over the bronchoscope is again

passed, this time to inspect the whole of the remaining bronchial tree and to clear it of any blood or secretions that may have accumulated, or spilled over, into it.

6. *The removal of foreign bodies*

Foreign bodies are most commonly inhaled by children and by idiots, who are apt to insert small objects such as peas, marbles or studs into their nostrils and so inhale them. Similarly nuts, coins, pins and loose teeth may be inhaled from the mouth during fits of choking or crying. Peanuts in particular are often inhaled by infants (to whom of course they ought never to be given); and although mothers are frequently mistaken in their belief that their child has 'breathed something in' it is safer when a peanut is in question to proceed with a bronchoscopy, even in the absence of any radiological change, rather than to run the risk of later impaction with bronchial obstruction, abscess or bronchiectasis, and the loss of a lobe (X-ray 6).

In adults the cause of inhalation of a foreign body, when it is not a tooth, is nearly always the dangerous practice of holding small screws, nuts, tacks or pins in the mouth while working (X-ray 7)

The symptoms produced by inhalation vary with the size and character of the object. When large, if it becomes impacted against the glottis itself or in the trachea, it will cause complete respiratory arrest and rapid death if it is not promptly removed. A patient should never be held head downwards in the hope that he will cough a large object free, for if it is lying in the trachea or one of the main bronchi it is then apt to be displaced and impacted against the under-surface of the glottis, converting a partial obstruction into a complete one with fatal consequences. A coin in the trachea usually lies in the sagittal plane, for it has to adopt this to pass between the vocal cords; whereas if it is in the oesophagus it lies transversely.

Solid foreign bodies which pass farther on are likely to cause partial or complete obstruction of the bronchus in which they lodge; and they often carry with them infection the effects of which vary with its severity. Clearly, extracted teeth or dental roots, fragments of tonsil or adenoid tissue are likely to be very septic, and if not swiftly removed will produce first segmental collapse of the lung distal to the impaction and then an acute putrid abscess in the collapsed segment. If the infection is less virulent, or obstruction incomplete, the presence of the foreign body may remain unsuspected for long periods, and the patient be quite unaware of what has happened. I recall a French lady, who was sent to be treated for bronchiectasis, in whom bronchograms showed involvement of the whole of her

middle and right lower lobes. Thinking the distribution a little unusual I bronchoscoped her in the presence of about a dozen students, telling them that such cases ought to be bronchoscoped in order to exclude a foreign body or some other unsuspected cause of the disease, and was both gratified and surprised to find one vertebra of a chicken impacted in her right main bronchus. Our teaching efforts are not often so well rewarded. The vertebra was exceedingly difficult to make out on a plain X ray and had not caused distal collapse because she had been able to go on breathing through its many interstices. Searching her memory she recalled choking a year before over an excellent pot-au feu. It should be noted that even such dense objects as teeth may be remarkably difficult to distinguish among the lung markings.

A similar instance was that of a mechanic who came merely with a troublesome but quite unproductive cough. Radioscopy showed a small nut in his right main bronchus. He had been holding the nut in his mouth while working four months previously. The hole in its centre had allowed aeration to continue and after its extraction the lung was found to be quite undamaged (X ray 7).

Radiographs should always be taken both in inspiration and expiration, for if the obstruction is only partial air may enter the lung as the bronchi distend on inspiration but be unable to escape again as they contract during expiration. As air is expelled the mediastinum is displaced to the unaffected side, and the diaphragm on the side with the blocked bronchus is unable to ascend. This is the condition of *obstructive emphysema* and is sometimes produced similarly by growths (X ray 6-62).

When it is radio-opaque a foreign body's exact location is plotted by postero-anterior and lateral films, and if possible its character, shape and position within the bronchus determined. The most suitable instrument for its removal is selected and bronchoscopy is carried out under the best possible conditions, for extraction at the first attempt is likely to be much easier than at a second. In the clinic of Chevalier Jackson it is customary to reduplicate the object and its position within the hose pipe bronchus of a dummy man so that a complete plan of campaign may be worked out, and if necessary a suitable instrument made or adapted.

If an object has been in position a long time oedema of the mucosa or granulations proximal to it not only constrict the lumen but may conceal the foreign body and attempts at removal are unsuccessful unless the swelling is first passed, and pressed back by expanding forceps. Conical bodies such as teeth are easily driven farther down

a bronchus if they are insecurely grasped at their presenting end with forceps; and it is better to pass an instrument beyond them and hook them back from their point of impaction.

Pins, especially open safety-pins, must be extracted with special care lest they pierce the bronchial wall during the process, and cause pneumothorax or empyema. If the point is directed upwards it is seized and drawn into the lumen of the bronchoscope; if downwards, the whole pin is removed with the point trailing, and if in an intermediate position it must first be manœuvred into one of the other two before removal is begun (X-ray 8).

Many foreign bodies are too large, or of an unsuitable shape, to be drawn up the bronchoscope. In that case they must be firmly secured with forceps and held close to the distal end of the instrument so that all can be withdrawn in one clean sweep, the object following through the glottis in an 'aftercoming' position.

As the right main bronchus more nearly continues the course of the trachea than does the left there is a natural tendency for foreign bodies to find their way into it rather than into the left if the patient is upright at the time of inhalation, as he usually is. Small objects will go farther, and enter one of the basal segments of the right lower lobe, and here may be quite beyond the reach of a normal bronchoscope. As a much smaller segment of lung is involved their consequences are not as a rule so serious, but if they cannot be extracted bronchoscopically thoracotomy may be necessary. The object can then be palpated (for it is always lying peripherally), and removed after an incision has been made directly down upon it through the narrow fringe of lung which covers it.

PULMONARY RESECTIONS

Preoperative Care

Emphasis upon various preoperative measures depends upon the pathological condition for which surgery is undertaken. In patients with bronchiectasis or other forms of lung sepsis preparation is chiefly directed to securing as much natural drainage as possible by posture and to antibiotic therapy. In the cancer age group the main concern is usually to improve respiratory efficiency by breathing exercises. Tuberculosis patients must have adequate bed rest, and streptomycin, to reduce disease activity to a minimum. These individual requirements, and the special problems occurring postoperatively, are separately discussed in the appropriate chapters. Certain preparations, however, are common to all.

1 *General nutrition* Patients whose appetites are poor or who have been losing weight, are often seriously deficient both in calory and vitamin intake, and in hydration. If time permits a suitable high calory diet supplemented by 400 mg. of ascorbic acid daily and by other vitamins in readily assimilable form is given. It is even more important to correct dehydration, and the patient should drink at least 2 litres of fluid daily until urinary chlorides are normal and the tongue is clean and moist.

2. *Oral sepsis* is common in Great Britain, where the standard of dental hygiene is the lowest in the world. The majority of those with teeth have dental caries. Most of the population over middle age is edentulous. The passage of bronchoscopes or intratracheal tubes through septic mouths is to be avoided. Loose teeth should be removed, scaling of others carried out, and pyorrhoea treated. Time seldom permits all carious teeth to be dealt with, and wholesale extractions are contra-indicated.

3 *Anaemia* contributes to dyspnoea and must be corrected, usually by transfusion. This should precede operation by at least three days. All resections are performed with an intravenous drip transfusion in progress so that blood loss is made good as it occurs. A minimum of two pints of cross-matched blood is generally required. Surgery should not be undertaken except in emergency until the haemoglobin level is above 80 per cent.

4 *Postural drainage* is indicated in all patients with purulent sputum

whether the pus is associated with bronchiectasis, or comes from a necrotic growth, or from beyond a partial bronchial obstruction. In the course of diagnosis the exact position of the lung lesion will have been ascertained, and the appropriate posture for gravitational drainage of the affected lobe or segment determined. The patient is encouraged to remain for as long as possible each day in this posture providing sputum is produced. Breathing exercises, forced coughing, and percussion (by a physiotherapist) of the area of the thorax related to the septic lung, increase the effectiveness of treatment (see 'Bronchiectasis', p 114)

5. *Breathing exercises* are carried out by all thoracic surgical patients before and after operation. Even young people who do not require much preoperative exercise learn to co-operate and practise the movements they will have to perform subsequently, while their chests are still intact and painless. Vital capacity estimations provide a measure of the success of exercises in improving mobility of the ribs and of the diaphragmatic excursion.

Chest movement is always decreased in the presence of pulmonary disease, and the more chronic the disease the greater the impairment. Ribs fall together, intercostal muscles and accessory muscles of respiration waste, the spine is flexed and rotated towards the affected side. Thus scoliosis and other postural defects are often present and must be corrected before normal respiratory movements can be resumed. Children are particularly prone to these deformities both before and after surgery.

Although respiration is performed unconsciously the respiratory muscles are voluntary and can with practice be voluntarily controlled. Thus patients are able, with skilled supervision, to learn dissociated chest movements and concentrate upon the mobility of one apex or base according to need.

Diaphragmatic movements are of great importance to those whose ribs have become fixed with age and to patients suffering from bronchospasm and difficulty in expiration. In many elderly people respiration depends almost wholly on the excursion of the diaphragm, so that phrenic paralysis is a serious disability. The abdominal muscles are accessories to diaphragmatic movement.

All breathing exercises are at first supervised and assisted by the physiotherapist, but later carried out frequently and independently. They are combined with exercises for the limbs and abdominal muscles, for all muscles waste, and joints become fixed, with disuse. Venous blood is returned from the lower extremities largely by muscle action. If this ceases blood stagnates, may clot, and so give

rise to pulmonary embolus. The less muscle wasting occurs, the shorter is convalescence likely to be.

6 *Antibiotics* All non tuberculous pulmonary resections are performed under penicillin cover and all tuberculous resections with streptomycin, unless the predominating organisms are resistant, in which case a suitable antibiotic is substituted. If no overt lung infection is present it is sufficient to begin penicillin in doses of 500 000 units a day 48 hours preoperatively and to continue it for the first postoperative week. Streptomycin is given in doses of 1 G a day (combined with P.A.S. 5 G six hourly) for not less than a month preoperatively and continued much longer according to need postoperatively.

Twice-daily injections of penicillin become very distressing to patients, and especially to children if they are prolonged. A single daily injection of procaine benzyl penicillin in oil is usually perfectly satisfactory and courses of antibiotics ought never to be unnecessarily protracted.

7 *Premedication* For an average adult male this usually consists of Omnopon gr $\frac{1}{2}$ and hyoscine gr $\frac{1}{160}$ injected one hour before operation. Patients over 65 should receive only half this dose of hyoscine, i.e. gr $\frac{1}{320}$.

Most technical difficulties encountered during pulmonary resections stem from three main causes

(1) Dense adherence of the lung to the chest wall. Although present in some degree whenever the pleurae have been chronically inflamed, this is seen most markedly in tuberculous patients especially those in whom tuberculous empyema is also present. The area of fusion is circumvented, and the diseased part of the lung left undisturbed, by dissection in the plane outside the parietal pleura, which is separated from the chest wall relatively easily and left attached to the lung. The process is attended by some shock and blood loss from the chest wall may be considerable unless carefully controlled by hot packs and coagulation. Fortunately the region of the hilum is usually comparatively free of gross adherence.

(2) Inflammatory reaction around the pulmonary vessels rendering their dissection and ligation difficult. These vessels are normally ensheathed in areolar tissue which becomes thickened and matted with inflammation. It is absolutely necessary to cut open these fibrous sheaths before the vessels and their branches can be defined and even in severely diseased cases the vessel will be found free within the sheath. Haemorrhage during dissection is usually caused by failure to carry out this step adequately.

(3) Extension of a lung neoplasm into neighbouring structures, including the hilum. Sufficient length of the pulmonary vessels must be left to ensure safe ligation, so that intrapericardial ligation is often required. Direct involvement of pericardium, diaphragm or ribs is dealt with by removal of these structures *en bloc* with the lung. It is sometimes possible also to resect part of the atrial wall by the application of finely toothed curved clamps and suture of the sectioned area of the myocardium. The dividing line between operable and inoperable growths depends on the skill and experience of the surgeon.

Thoracotomy

Thoracotomy for pulmonary resection is performed with the patient either in lateral or prone position on the operating table (Figs. 9, 10). The approach chosen depends on whether or not an intrabronchial blocker is employed by the anaesthetist. Such a blocker is contra-indicated (a) in young children, for their bronchi are too small; (b)

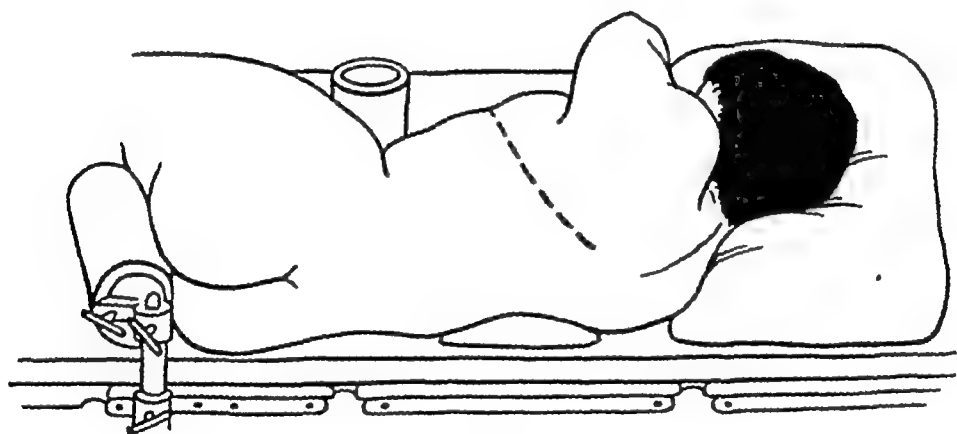


FIG 9

The usual position of the operating table for postero-lateral thoracotomy. The patient's hips are firmly held by special supports and the uppermost arm lies freely over the far side of the body.

in patients who have bilateral suppuration; (c) when a broncho-pleural fistula is present, and (d) whenever there is serious risk of spillover of secretions from the diseased into the healthy lung. In all these circumstances in which a blocker is unsuitable the prone, or 'face-down' position is used. The patient lies upon flat pelvic and upper chest rests arranged to secure gravity drainage of both lungs up the bronchial tree so that secretions are easily aspirated from the

trachea (Fig. 10) The thoracotomy side lies slightly overlapping the table edge, and the arm on this side hangs vertically downwards. The *advantages* of this position are (1) complete safety from spill over (2) avoidance of intrabronchial blockers, and (3) the surgeon can operate seated. The *disadvantages* are (1) some loss of freedom of access to the hilar structures (2) cardiac pulsation is more prominent, for the lung lies on the pericardium and (3) infants with soft chest walls sometimes do not tolerate the position well before the thorax is opened and just after it is closed. Prompt entry and exit are therefore indicated.

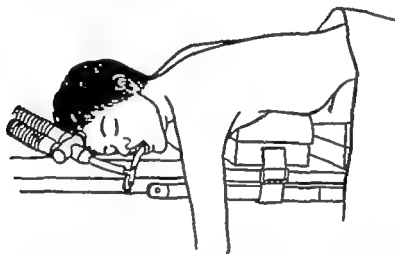


FIG 10

The prone position for 'face-down' thoracotomy in which no intrabronchial blocker is employed, secretions from the bronchial tree draining gravitationally towards the mouth. This position is always employed for pulmonary resection in children, or in patients with bilateral suppuration.

In most adults the lateral position is used unless one of the contra indications mentioned above exists. The patient lies with the back at right angles to the surface of the table and close to the edge nearest the surgeon. An L-shaped, sponge-rubber covered rest supports the dependent axilla, gently arching the chest and spreading the upper most ribs. The upper arm hangs loosely over the far side of the table and is used for intravenous therapy. Two other vertical supports one in front against the pelvis and one behind the knee flexure, fix the body and allow the whole table to be tilted towards the surgeon if required (Fig. 9).

Whichever position on the table is preferred the method of entering the thorax is the same. The incision passes obliquely round the

angle of the scapula from about an inch lateral to the vertebral spines behind to the costal cartilages in front and follows roughly the line of the sixth rib. This line, and two or three intercostal nerve roots above and below it, are infiltrated with a solution of 0.3 per cent Xylocaine (or 0.25 per cent. Procaine) to which 1 cc of 1 : 1000 adrenaline is added just before use. This much reduces bleeding, and the amount of general anaesthesia required. After the skin incision the latissimus dorsi is divided, exposing the angle of the scapula and

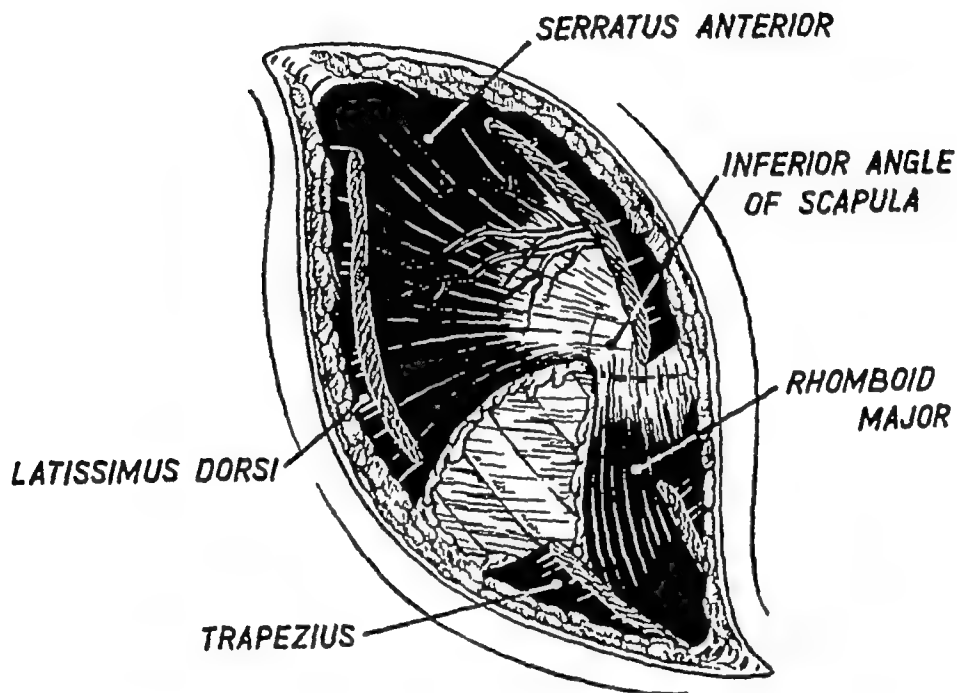


FIG 11

A standard left postero-lateral thoracotomy wound after division of the skin and the first muscle layer.

the triangle of fatty areolar tissue between the anterior edge of the trapezius behind and the posterior border of serratus anterior in front (see Fig 11). This triangle is opened, and both muscles cut to lay bare the ribs. The anterior portion of the serratus is divided in the direction of its fibres, and it is often necessary to split the inferior edge of rhomboideus major posteriorly. All bleeding points are coagulated. Most of the vessels lie on the fascial sheaths of the muscles and can be secured before they are cut.

The scapula is now retracted and the ribs counted down from the origin of serratus anterior from the second rib. For standard pneu-

monectomy or lobectomy best access is gained by resection of the whole length, from rib neck to costal cartilage of the sixth rib. The periosteum is incised longitudinally by diathermy and stripped from the rib with a curved elevator in two clean sweeps—from back to front along the upper border of the rib and from front to back along the lower. The rib is divided at its neck and costochondral joint by a guillotine costotome (see Fig. 12). The periosteal bed is incised, and the pleural space carefully entered. If it is desired to cover the bronchial stump later with an intercostal muscle graft, this is next cut incorporating the intercostal artery, vein and nerve of the resected rib but leaving enough muscle on the upper edge of the seventh rib to make subsequent closure easy.

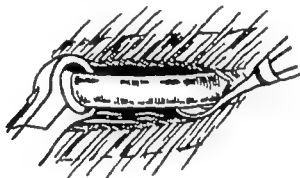


FIG 12

Method of rib resection. The periosteum has been stripped (in this case only from a short segment of rib) with a periosteal elevator. The intercostal vessels and nerves fall away with the lower leaf of periosteum. A guillotine costotome divides the rib. Note the direction of the fibres of the external intercostal muscles.

For operations in which an intercostal graft is not necessary (i.e. segmental resections) there is often no need to resect a rib and entry into the pleural cavity is gained by incising the periosteum after it has been stripped from the upper surface of the rib. This avoids the bleeding which attends division of the intercostal muscles themselves.

After wound cloths have been inserted the pleural cavity is widely exposed with the use of a Finochietto rib-spreader. Care is taken to divide adhesions between lung and chest wall as they are brought into view and to increase the spread slowly so that ribs are not broken. The freed lung is finally inspected, and if a growth is present its operability and the scope of the operation is determined.

PULMONARY RESECTIONS

Pneumonectomy

1. *Left* (see Fig 13) The pleural reflection round the hilum is cut, and the pulmonary ligament (from the diaphragm to the inferior pulmonary vein) divided. The inferior vein is defined, cleaned of all pericardial reflections, and a pneumonectomy or O'Shaughnessy

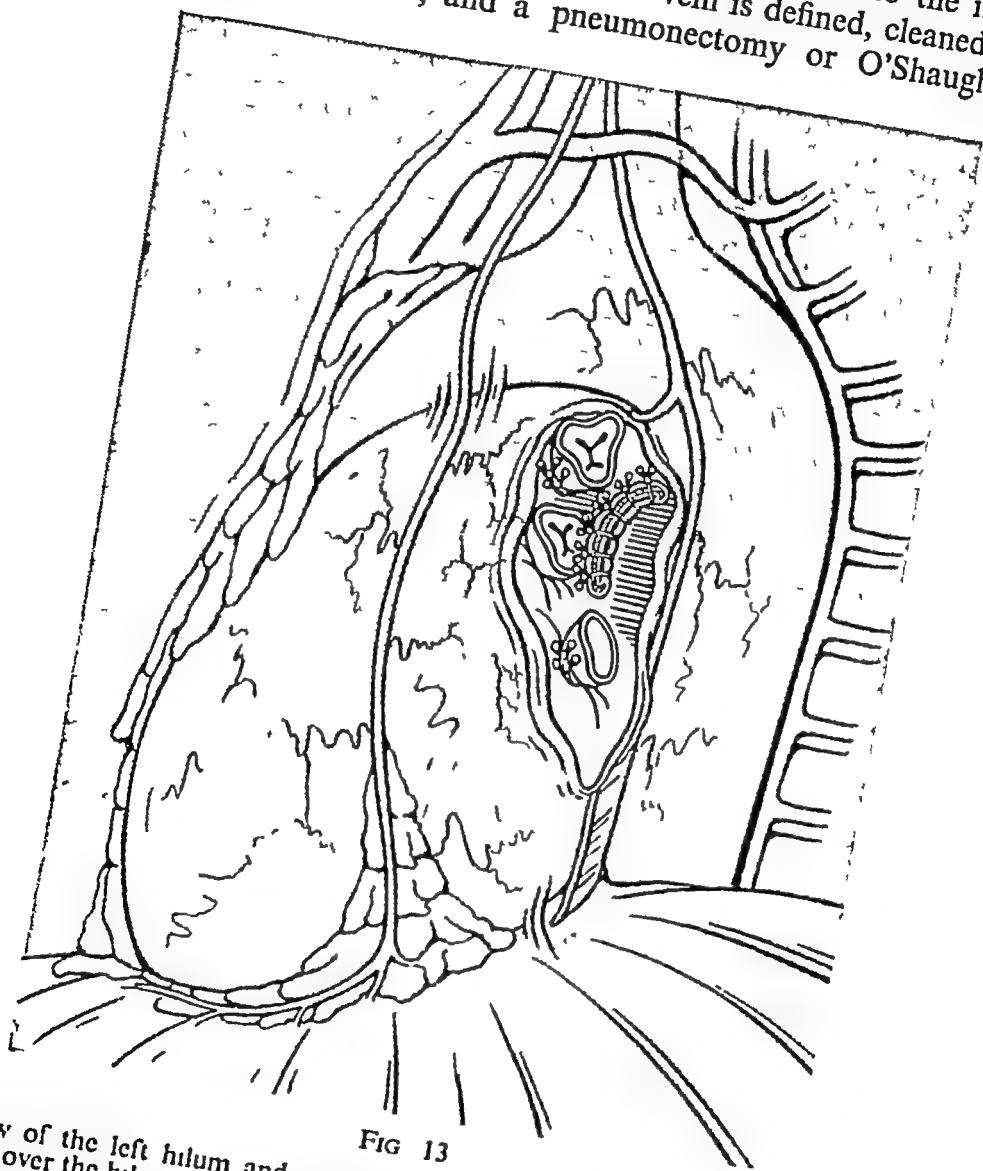


FIG 13

View of the left hilum and mediastinum after pneumonectomy. The aorta arches over the hilum and is crossed by the phrenic nerve in front and the vagus behind, the latter giving off its recurrent laryngeal branch which hooks round the ligamentum arteriosum to run up behind the aorta to the neck. Just below the arch is the pulmonary artery, with the bronchus below and behind it. Below the artery, and in front of the bronchus, is the superior pulmonary vein; below it the inferior pulmonary vein.

forceps passed round it to secure a 25-gauge linen thread delivered in bow string fashion (Fig. 14a) The vessel is doubly ligated as close to the heart as possible tied distally (or its branches secured) near the lung, and cut between these ligatures to leave a sound cuff on the cardiac side. If necessary the stump may be transfixed. Divi

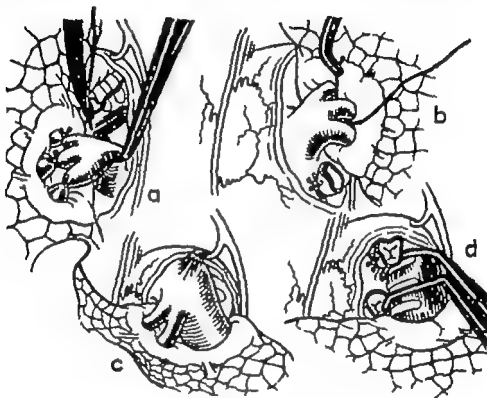


FIG 14 Left pneumonectomy

- (a) Ligation of the inferior pulmonary vein.
- (b) The inferior vein has been divided and the branches of the superior pulmonary vein are now secured.
- (c) The pulmonary artery emerging from the pericardium. Note the ligamentum arteriosum round which the recurrent laryngeal nerve loops.
- (d) After division of the artery a light curved clamp controls the bronchus proximally a toothed one is applied on the lung side, and the bronchus is cut between them.

tion of the pulmonary ligament and inferior pulmonary vein mobilizes the lung and makes subsequent procedures safer for access is afforded to the back of the superior vein and to the bronchus and the whole pedicle of the lung is readily controlled. The lung is drawn posteriorly with Duval forceps and the superior pulmonary vein is next similarly cleaned, ligated and divided (Fig 14b) (It is better to

PULMONARY RESECTIONS

Three holes are now drilled at intervals along the seventh rib, and pericostal sutures of doubled No. 4 chromic catgut are passed through them and round the fifth rib above. This avoids any risk of compressing the seventh intercostal nerve. A rib approximator draws the two ribs together and is removed after the sutures have been tied. The

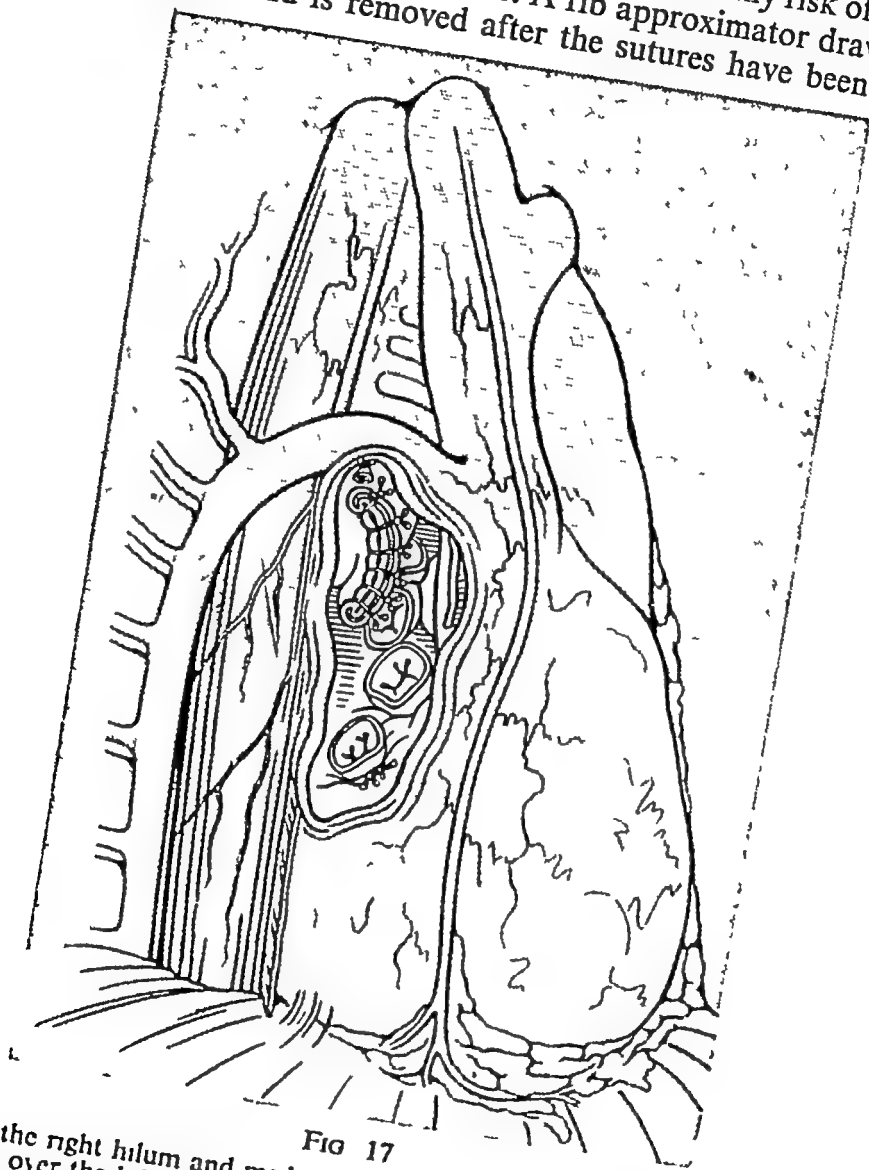


FIG 17

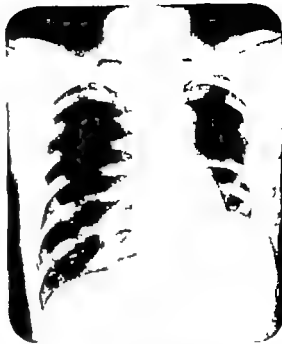
View of the right hilum and mediastinum after pneumonectomy. The azygos vein arches over the hilum as it leaves the superior vena cava to run down the posterior mediastinum beside the oesophagus. The phrenic nerve lies first on the superior vena cava, then on the pericardium. Immediately anterior to the amputated bronchus is the pulmonary artery with the superior pulmonary vein just below and a little in front of it, and the inferior pulmonary vein at the lowest point of the hilum. If pneumonectomy is performed for carcinoma, the azygos vein is divided.



9

X-ray 9 After pneumonectomy pleural space usually fills with serum and effusion. Here left lung had been removed. Trachea and heart are displaced to the left. Left dome of diaphragm has risen, ribs have fallen in and become more sloping than those on opposite side. These things help reduce space left.

X-ray 10. Following a left pneumonectomy no serum has accumulated in the empty pleural space and the patient consequently needs periodic air refills. An alternative to these would be a left lateral thoracoplasty. Note regeneration of the resected 7th rib.



10



11



12

X-ray 11 The left lower lobe has just been removed (see X-rays 2 and 12), and postoperative collapse has occurred of the left upper lobe which is now opaque because it is airless. Immediate bronchoscopy is indicated.

X-ray 12. After bronchoscopy to relieve postoperative atelectasis seen in X-ray 11 the upper lobe has re-aerated. Atrial convexity is now seen to right of mid-line, in spite of the fact that left lower lobe has been removed (compare with X-ray 2) because pressure in left pleural cavity is normal again.



13



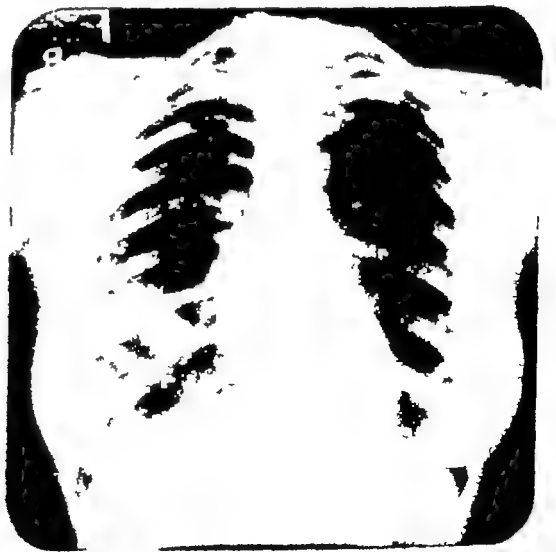
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X-ray 13 A left spontaneous haemopneumothorax. Note the displacement of the mediastinum to the opposite side. Bleeding was severe and thoracotomy required to coagulate the torn adhesion responsible, evacuate clotted blood, and re-expand lung.

X-ray 14 A right tension pneumothorax due to air leakage following the excision of emphysematous bullae seen in X-ray 37. Note mediastinal displacement and compression of the opposite lung. After increased suction on the drainage tube the right lung re-expanded fully. (See X-ray 38.)



15



16

X-ray 15 Spontaneous left pneumothorax. The heart is displaced to the right, the left lung is airless and compressed against the mediastinum. No lung markings whatever are visible in the left intercostal spaces. This air leak was due to a ruptured bleb at the left apex.

X-ray 16 A chondroma deforming the anterior end of the right 5th rib.

intervening intercostal muscles are sewn up with a continuous No. 2 chromic catgut suture and the other muscle layers are similarly closed. Interrupted nylon is used for the skin and subcutaneous layers. Drainage is not required after pneumonectomy, but when the dressing is in place the rests removed, and the patient on his back, the intrapleural pressures of the operated side are measured with a Morland needle and Maxwell box and enough air is added or withdrawn to make the pressures atmospheric (e.g. a swing of +10 to -10 cm. of the manometer scale).

2. *Right* (see Fig. 17) This differs from the operation on the left in minor anatomical points only. The pulmonary ligament and inferior pulmonary vein are dealt with as before. The pulmonary artery instead of entering the hilum in one large trunk, bifurcates into a smaller upper and much larger lower division just under cover of the superior vena cava and the lower of these divisions lies almost wholly behind the superior pulmonary vein (Fig. 18a). It simplifies dissection, and therefore contributes to safety to ligate and divide the vein first. It will then be seen that a fan-shaped fibrous reflection of pericardium overlies the base of the artery, and is attached to a lymphatic gland lying at the bifurcation of the main trunk into upper and lower divisions. This fold must be lifted from the arterial wall by forceps passed beneath it, and cut, thus laying bare the main arterial trunk (Fig. 18b). As on the left, the artery is freed from its bed by the index finger and thumb ligatures are passed round it and tied as proximally as possible. Each of the two divisions is separately secured distally (Fig. 18c).

As the right bronchus is shorter than the left and does not lie beneath the aortic arch it is easier to clamp and divide at its most proximal point. A large constant bronchial artery lies on its posterior aspect (Fig. 18d). The carinal lymph glands are removed with the lung as before.

The azygos vein is divided between ligatures and the lateral and pretracheal lymph glands together with the fatty tissue surrounding them dissected. The pretracheal glands lie in a fossa limited by the trachea behind, the superior vena cava in front and the aorta on the left.

3. *Intrapericardial pneumonectomy* When a growth involves the pericardium, or makes extrapericardial ligation difficult or hazardous the pericardium is widely opened behind the line of the phrenic nerve, and the pulmonary veins secured and tied on the atrial wall itself. On the left side the ligamentum arteriosum is cut and the pericardium dissected from the artery so that it may be ligated close to

its origin. The pericardium behind the lung hilum is similarly opened and the whole involved area removed with the lung. This procedure

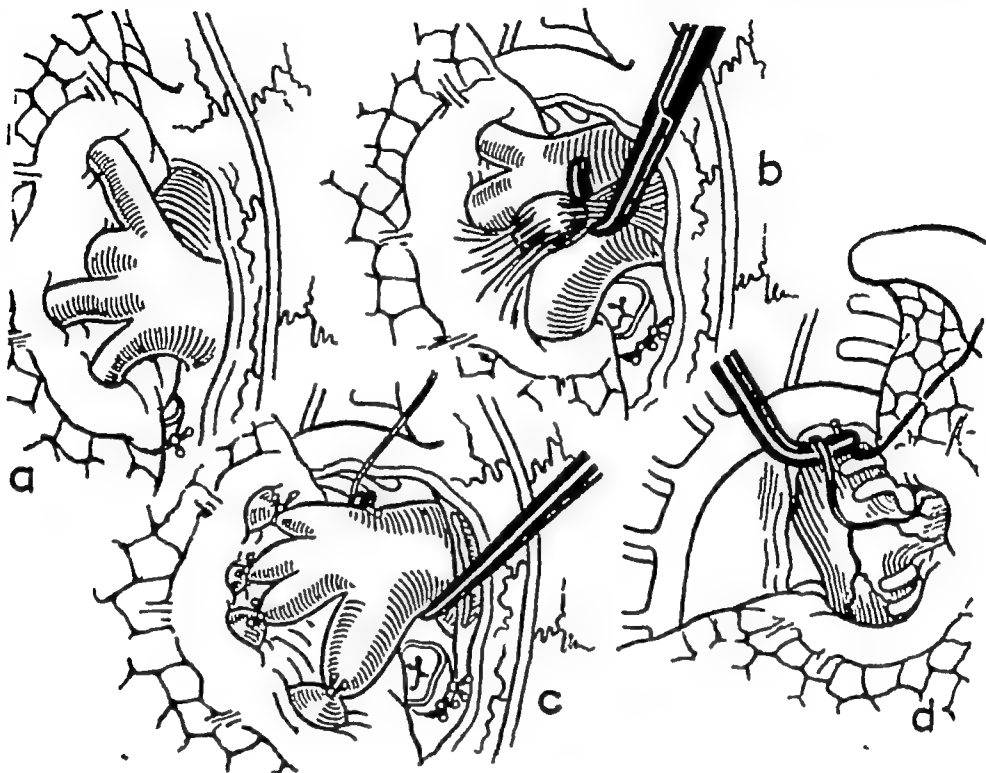


FIG 18. Right pneumonectomy

(a) The branches of the superior pulmonary vein from above down are, apical, posterior and anterior segmental branches, and the branch to the middle lobe. Note how the lower division of the pulmonary artery lies immediately behind the vein

(b) The pericardial fold overlying the pulmonary artery and attached to its bifurcation is lifted and cut

(c) After division of the fold both trunks of the artery are secured

(d) Ligation of a bronchial artery before division of the bronchus

greatly simplifies the operation, but is attended by a much higher incidence of atrial fibrillation than extrapericardial ligation.

Sleeve resections

Sometimes growth spreads from an upper lobe bronchus on to the main bronchial wall so that removal of it by lobectomy is impossible. Pneumonectomy however may be contra-indicated in a patient already breathless, especially if it is the right lung which is affected. This is especially so if the neoplasm is an innocent bronchial adenoma. In such cases, it is sometimes possible to remove the affected lobe

and bronchus by dividing the main bronchus above and below the growth securing the vessels supplying the upper lobe, but leaving those to the rest of the lung intact. The two cut ends of the main bronchus are subsequently sutured together and healing is usually uneventful.

Lobectomies

1 *Left upper lobectomy*

The superior pulmonary vein is first ligated. The pleural reflection over the main pulmonary artery is cut, and the surface of the vessel thoroughly cleaned. The sheath of tissue in which it lies must be

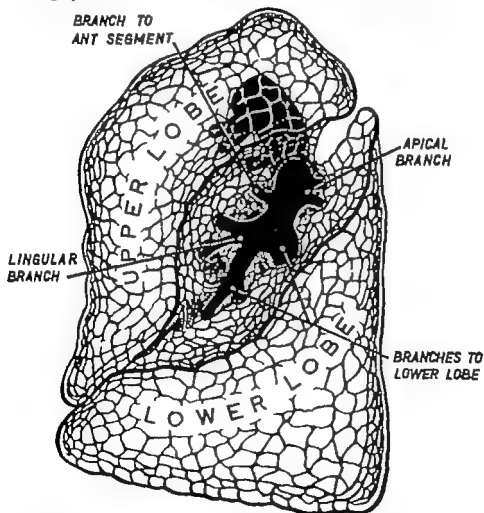


FIG 19

The interlobar fissure of the left lung has been opened to display the lobar and segmental branches of the pulmonary artery

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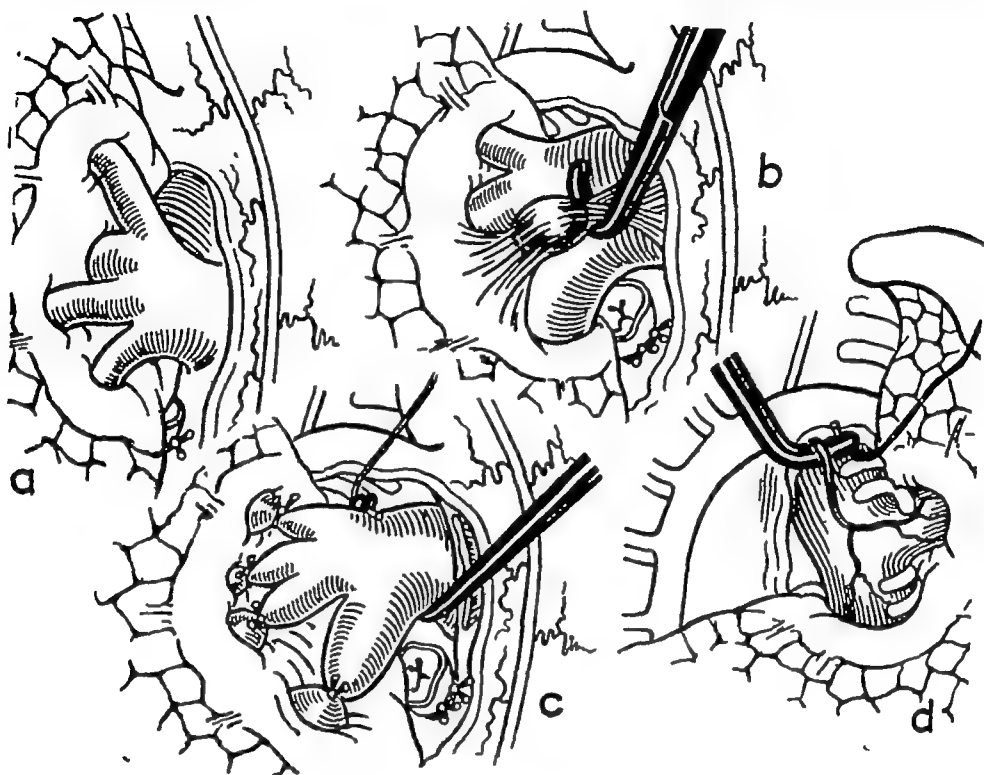


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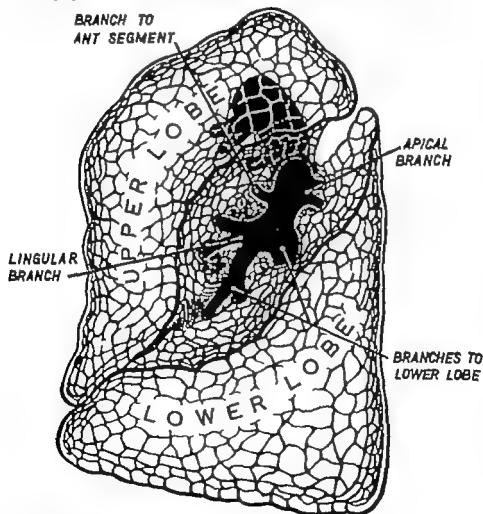


FIG 19

The interlobar fissure of the left lung has been opened to display the lobar and segmental branches of the pulmonary artery

opened and stripped off. Failure to do this is the cause of most subsequent difficulties. Further dissection is then relatively easy and the branches of the artery to the upper lobe segments can readily be identified, ligated and divided (see Fig. 19). A sufficient length of each is freed to provide an adequate cuff; and as the vessels are themselves thin-walled and often friable care is taken to avoid excessive traction on the lung. For this reason also it is safer to tie their distal extremities before placing the proximal ligature. As these branches are tied in succession, the main artery is followed round the hilum of the lung into the interlobar fissure where the branches to the anterior and lingular segment are usually found (Fig. 19).

The upper lobe bronchus is cleared and the small bronchial arteries accompanying it ligated or undersewn. When the bronchus has been thoroughly defined down to its origin from the stem bronchus clamps are lightly applied close to this point and the bronchus cut between them. Sufficient flange is left for subsequent closure of the stump with fine interrupted linen sutures. After withdrawal of the intrabronchial blocker, saline is run in to cover the stump and it is tested for air leaks. It is then covered by an intercostal muscle graft sewn in place over it, and passing to it beneath the main pulmonary artery. Two water-sealed drains (Fig. 22) are left in the chest, one draining effusion from the base of the pleural cavity, and a narrower one for the removal of air from the apex. Both are connected to pumps which maintain a negative suction of about 40 cm. of water.

2. Left lower lobectomy

The pulmonary ligament is cut, and the inferior pulmonary vein defined and cleaned. Its position is marked by a small lymphatic gland found constantly at its inferior border. After the vein has been doubly ligated and cut, the interlobar fissure is opened and dissected down until the pulmonary artery is found lying in its depths (Fig. 19). Its branches to the lower lobe, usually comprising two main trunks and a smaller vessel to the apical segment, are tied and divided. A bronchial artery, always closely attached to the anterior surface of the bronchus (which is now exposed) is under-run by a suture. The rest of the bronchus is cleared, divided and closed. It is sufficient to leave one basal water-sealed drain in position before closing the chest.

3. Right upper lobectomy

The superior pulmonary vein is dissected clear, and the part of it supplying the upper lobe ligated. Care is taken not to damage or

occlude its branch to the middle lobe. The pulmonary artery is defined as in a pneumonectomy and the smaller of its two main trunks that supplying the apical and anterior segments of the upper lobe, ligated and cut. A lymph gland is constantly found in the main bifurcation of the pulmonary artery and must be cleaned off the remaining arterial trunk which runs down to enter the fissure and supply the other lobes. The branch to the posterior segment of the upper lobe is sometimes found leaving the main artery just beyond the bifurcation, but usually it does not do so until the fissure is reached, when it courses to its segment parallel with, and just inferior to the upper lobe bronchus (Fig. 20) The vessel may either be secured by opening

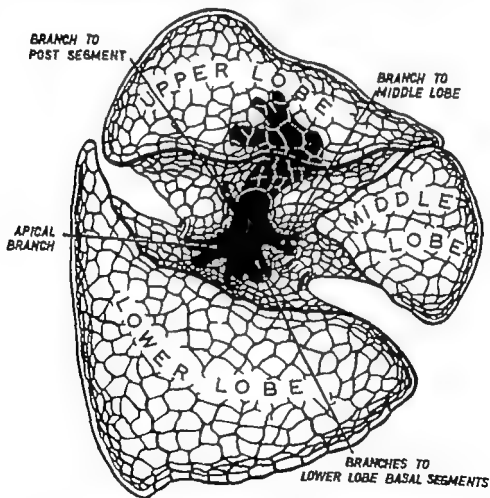


FIG. 20

The interlobar fissures of the right lung have been opened to display the lobar and segmental branches of the pulmonary artery

the interlobar fissure, or after division of the bronchus itself. The bronchial stump is closed as before, and the upper lobe peeled from the inflated middle lobe. The fissure between the two is incomplete and care is taken not to damage the middle lobe artery which is closely related to the ligated stump of the superior pulmonary vein. Upper and lower water-sealed drains are left in place at the end of the operation.

4 *Middle lobectomy*

The middle lobe branch of the superior pulmonary vein is identified and cut between ligatures. Occasionally it enters the pericardium as an independent vessel, or less often flows into the inferior pulmonary vein. The interlobar fissure is opened at the apex of the middle lobe, and the main arterial trunk to the lower lobe dissected clear (Fig 20). As this vessel is followed upwards its middle lobe branch comes into view. In about half the cases there are two branches, one just above the other. After ligation of these, the bronchus is divided and the lobe removed. One basal drain is employed.

5. *Right lower lobectomy*

The pulmonary ligament is divided and the inferior pulmonary vein ligated and cut. The fissure is opened and the branches of the artery to the lower lobe identified and secured (Fig 20). Care is taken not to injure the middle lobe branch. The artery to the apical segment often arises well above this level on the opposite side of the main trunk, and may have to be separately ligated. One basal drain is left in place.

Segmental resections

Healthy lung must be preserved whenever possible. It is often possible in the treatment of pulmonary tuberculosis and bronchiectasis to remove diseased areas by segmental resection alone. The procedure is similar for all the segments. First the branches of the pulmonary artery supplying the lobe to which the diseased segment belongs are carefully defined, and the one running to the segment itself is identified and secured. Immediately deep to this vessel lies the segmental bronchus. After it has been freed it is lightly clamped while the anaesthetist inflates the rest of the lung to make sure that the whole diseased area is included. The segment is then stripped off its neighbours by a combination of traction and blunt dissection (Fig 21). The tributaries from the segment to the pulmonary vein are tied as they are encountered. On the left side the apical and

posterior segments of the upper lobe share a common bronchus and artery for a short distance and may therefore have to be removed together. If dissection is carried sufficiently far distally however, it is usually possible to separate them. The lingular segment of the left upper lobe is very commonly involved in bronchiectasis, and must therefore often be resected, either by itself or together with the



FIG. 21

Segmental resection of the posterior segment of the right upper lobe. The clamped and divided bronchus to the segment is being drawn away from the lung peeling the segment from its neighbours on whose raw surfaces the intersegmental veins can be seen. The apical and anterior segmental bronchi of the upper lobe are intact. Between them lies the ligated stump of the posterior segmental vein, below the anterior branch the cuff of the posterior segmental artery which in this case has left the main trunk in the interlobar fissure.

left lower lobe. Its artery is secured in the interlobar fissure, its vein is readily accessible where it joins the superior pulmonary vein close to the pericardium, and its bronchus, dividing into two branches, lies between artery and vein.

Apart from the preservation of healthy lung, a great advantage of segmental resection in tuberculous patients is that it often avoids the need for subsequent apical thoracoplasty to obliterate dead space

and prevent overstretching of the lung left behind. Its only disadvantage is that when chronic disease has been present adjacent segments may be affected by fibrosis and emphysema, so that troublesome air-leaks occur from their raw surfaces. Every care is taken therefore at the time of operation to close bronchiolar leaks by undersewing them, but the finer alveolar leakage is most effectively stopped by securing prompt re-expansion of the lung and its adherence to the chest wall as rapidly as possible. This is achieved by continuous postoperative suction upon two drainage tubes, one dependent to remove fluid from the pleural cavity, one apical to remove air.

Postoperative care and complications

At the conclusion of any type of pulmonary resection, and before the patient leaves the operating table, bronchoscopy is performed and blood or secretions carefully aspirated from both sides of the bronchial tree.

The patient returns to the ward wearing a light, well-fitting, plastic oxygen mask and with the intravenous drip still running. A mask that does not fit or is not comfortable is worse than useless. Oxygen is not usually required for very long after consciousness is regained, but if any degree of cyanosis persists it is continued, and it should be available at the bedside at all times. Elderly emphysematous patients may hover on the borderline of anoxia in the first few days after resection for carcinoma, and oxygen lack sometimes induces mental disturbances such as agitation, depression, or even suicidal tendencies. These are relieved by the administration of oxygen.

As well as the measures usually employed to combat shock after any major operation, care is taken to see that a sudden fall in blood pressure does not occur in the first few hours after the patient's return to bed. If an extensive extrapleural strip has been performed there may be reactionary oozing from the chest wall. A considerable volume of blood may gradually be lost in this way so that additional transfusion and aspiration are required. If a drainage tube has been employed such bleeding is at once evident, otherwise it is diagnosed by the usual signs of postoperative haemorrhage. Chest wall oozing of this kind does not usually persist. The treatment of *continued* bleeding is securing the responsible vessels. Blood transfusion is supplementary to this, not an alternative. Bleeding is more likely to occur from a systemic vessel, such as a bronchial or intercostal artery, than from a pulmonary one in which tension is much lower; but if a ligature should slip from a large pulmonary vessel, the resulting

haemorrhage would be swift and catastrophic. Major trunks must therefore be doubly ligated and safe cuffs left beyond the ligatures. If any doubt as to the adequacy of a cuff exists the vessel is transfixed.

The aim of all postoperative treatment is the restoration as quickly as possible, of normal function. Normal respiratory function depends upon two things, full aeration of the lung and full mobility of the chest wall and diaphragm. Whatever interferes with these things must be combated: the patient must cough regularly to expel secretions in the bronchial tree and maintain full aeration of the lung. Dehydration makes secretions thick and difficult to cough up; therefore it is not allowed to occur. Morphine and other analgesics are given to subdue pain but not the cough reflex, and the head and neck are manually supported whenever a coughing effort is made. Although regular coughing and breathing exercises are insisted upon during waking hours the patient must not be exhausted, or awakened from sleep to perform them. Constricting bandages, and especially any encircling the chest, are banned. Inability to cough and the accumulation of bronchial secretions, call for bronchoscopic aspiration before atelectasis occurs. This is particularly urgent when pneumonectomy has been performed for the patient has no margin of functioning lung to spare. Antibiotic cover is normally continued for the first postoperative week, but purulent sputum or any other evidence of infection calls for further therapy.

After pneumonectomy it is of prime importance to keep the mediastinum central and so prevent either compression of the remaining lung by mediastinal shift towards it, or overstretching of the lung by a shift away from it. At the end of the operation after the patient has been turned on his back, the pressures in the empty pleural space are measured by a Morland's needle inserted into it and connected with the manometer of an artificial pneumothorax apparatus (usually a Maxwell box). Air is added or withdrawn until the manometer needle swings with respiration an equal distance on either side of the zero mark (e.g. +10 to -10 cm. of water) indicating that the pressure within the chest is atmospheric. During the next few days air may be absorbed from the cavity, or coughed into the soft tissues of the chest as *surgical emphysema* so that the intra-pleural pressure becomes negative and the mediastinum is drawn across or serum and exudate collect in the cavity raise the pressure and push the mediastinum away. In either case the position of the mediastinum must be corrected, and atmospheric pressure restored. It is necessary to carry out these adjustments every few days at first,

and respiratory embarrassment or discomfort in the early post-operative phase calls for verification of the intrapleural pressures. By the end of the first week enough fluid has usually accumulated in the space to maintain the mediastinum in a central position, and in most cases the space eventually fills completely so that further air refills are not required (X-ray 9). The diaphragm rises, the ribs fall in somewhat, and the mediastinum is moderately displaced to the side of operation. In a few patients very little fluid accumulates and the pleural space remains empty. Under these circumstances monthly refills of air are required in patients who have had pneumonectomy for cancer (X-ray 10). If at the end of two years refills are still needed the patient is offered the alternative of lateral thoracoplasty. After pneumonectomy for non-malignant conditions, such as tuberculosis or bronchiectasis, it is usual to perform a lateral thoracoplasty a month or six weeks postoperatively.

If fluid accumulates too fast in the pleural cavity it may cause cardiac embarrassment as well as mediastinal displacement and must be aspirated. It is in any case good practice to keep the fluid level below that of the bronchial stump for the first week lest a bronchial fistula (*vide infra*) develop.

Cardiac irregularities are common after pneumonectomy, especially in patients of the higher age group. Atrial fibrillation is most often seen and may last only for a few hours before normal rhythm is resumed. In severer cases prompt digitalization is necessary with 1 mg of intravenous digoxin, followed in four hours by a further 0.5 mg, and 0.25 mg thereafter four-hourly by mouth until the pulse rate falls to 70. Atrial flutter is less common and calls for electrocardiographic confirmation, as do rarer disorders such as nodal tachycardia.

The most serious postoperative complication of pneumonectomy is *bronchopleural fistula*. Before the advent of antibiotics pulmonary resections were attended by a prohibitively high incidence of fistula, leading sometimes to fatal aspiration of fluid from the side of operation into the remaining lung, but inevitably to contamination of the pleural cavity and consequent empyema. Failure of the bronchial stump to heal was the main impediment to the advance of chest surgery, and it was not until streptomycin became available that pulmonary tuberculosis could be treated by resection. Although bronchial fistula is now a rare disaster, it is still to be feared and avoided, for its consequences are always serious and it is difficult to treat.

The bronchus is amputated in such a fashion that no blind stump

is left in which an infected puddle of mucus can collect (Fig. 15) Section of the bronchus is never performed through an area of its wall affected by tuberculosis or involved by growth The proximal part of the bronchus is clamped lightly and not for long, lest its blood supply be interfered with, and meticulous care is taken in suturing the stump and in testing it for leaks afterwards Finally it is covered, preferably with an intercostal muscle graft, but failing that with a pleural flap

In non tuberculous patients bronchopleural fistula is most likely to occur during the first week or ten days but after resections for tuberculosis the risk remains for much longer and fistula formation tends to be late Resistance to antibiotics greatly increases this hazard.

Development of a bronchial fistula is usually signalled by a persistent cough, slight haemoptysis, a rise of temperature and eventually the expectoration of some of the serous fluid, stained brown by changed blood from the pleural cavity If the fistula is large, involving most of the bronchial stump there is seldom doubt about the diagnosis but if it is small its existence can be proved decisively by measuring the intrapleural pressures on the affected side. When a small fistula is present the pressure is usually atmospheric or slightly positive. (Occasionally the bronchial air leak is valvular so that an increasingly positive pressure is built up resulting in *tension pneumothorax* and calling for urgent relief) After the pressures have been read 300-400 cc. of air are withdrawn, and new measurements made over a period of five minutes or so to see whether a negative pressure is maintained. If it is not, the presence of a leak is proven, and the speed with which the pressure returns to atmospheric provides a measure of its size Methylene blue may also be injected into the pleural space and a watch kept for its reappearance in the patient's sputum pot.

The diagnosis of bronchopleural fistula calls for prompt action. The patient is warned not to lie on the side of his intact lung, so that fluid on the opposite side is kept away from the leaking bronchus until it can be completely aspirated. The withdrawn fluid is cultured, and if necessary a change made in the covering antibiotics, some of which are also injected into the space in the hope of sterilizing it. If break-down of the bronchus has occurred early, the best treatment is to reopen the chest at once identify the leak and sew it up anew but if much time has elapsed and infection is established, the chances of success are very poor Occasionally a small leak may heal spontaneously with the aid of antibiotics, and this is more likely if a

muscle graft has been employed. In most pneumonectomy patients it is necessary to resect a rib and insert a dependent drainage tube as for an empyema. This is likely to be a permanency.

After lobectomy or segmental resection the pleural cavity is always drained. If the whole, or part, of an upper lobe has been removed two tubes are employed, one reaching to the apex of the chest to

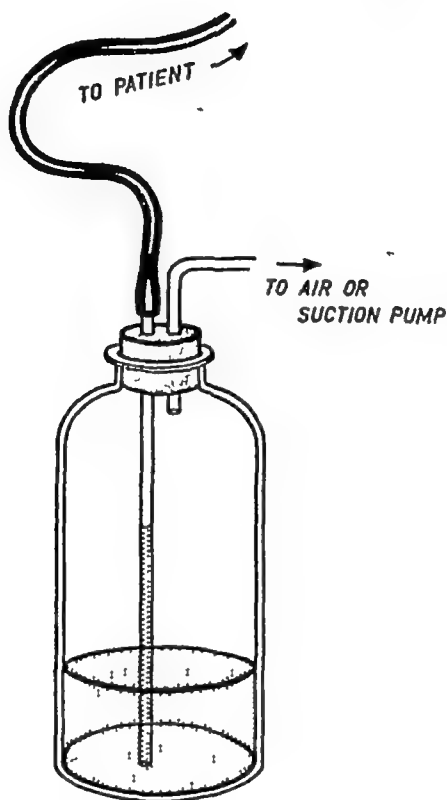


FIG. 22

A water-sealed drainage bottle. The intrapleural drainage tube is carried down under a measured quantity of saline or antiseptic solution and the negative pressure within the chest causes the fluid to rise up the tube in a meniscus which swings with respiration. The open-ended tube may be connected with a suction pump.

remove air, and a basal one to drain off fluid. Both are water-sealed (Fig. 22), and connected with Roberts' pumps exerting a continuous suction of 40 to 50 cc of water. The object is not only to remove impediments to full expansion of the lung left behind but also to seal off parenchymal air-leaks from raw areas by bringing them quickly into apposition with the chest wall. Usually the drainage tubes can be removed in 24 or 48 hours if portable radiography con-

firm full aeration of the lung. Expansion of the lung, and expulsion of air and fluid from the pleural cavity is promoted by regular coughing and deep breathing. If the pumps are stopped, the negative pressure in the pleural space is indicated by the fluid meniscus which rises and falls in the glass drainage tube with respiration, and if this free excursion ceases the tube has become blocked and must be cleared. Sometimes obstruction is due to blood clot which can be milked down the tube or expressed from its inner end, or to kinking of the tube at the patient's back rest, or by a misplaced safety pin on the bed-side but if blockage is permanent, or the swing ceases because the lung itself has fully expanded and sealed off the end, the tube should be removed, and residual air or fluid aspirated with a Martin's syringe. It is essential that air should not be allowed to enter the chest as the drainage tube is withdrawn. Therefore, after the retaining suture has been cut, the skin on both sides of the tube is pinched between finger and thumb the patient exhales and holds his breath and the tube is smoothly and quickly pulled out. A *tulle gras* dressing is firmly strapped over the closed wound.

By far the commonest complication of lobectomy is *atelectasis* of the lung left behind on the same side and this is caused by failure to expel secretions from the bronchus by coughing (X ray 11). Once bronchial obstruction has occurred, air in the lung distal to it is rapidly absorbed, and no coughing effort can possibly be effective in dislodging the mucous plug, for air in the lung is the charge which normally drives the pellet out. Instead a negative peripheral drag tends to draw the obstruction farther and farther out into the lung so that with every hour that elapses relief becomes more difficult. Apart from the serious loss of function, such a collapsed lobe will certainly become bronchiectatic if it is not re-expanded, and so long as collapse persists antibiotic cover must be continued. The incidence of postoperative atelectasis is greatest in children and in patients who do not make regular efforts to cough. It is higher in wards with poor nursing than in those with well-trained staff. It is more likely to occur if secretions become sticky because the patient is dehydrated and it is commonest on the second or third postoperative day when oedema at the bronchial suture line is greatest.

It is diagnosed by radiographic evidence of opaque (i.e. non-aerated) lung (X ray 11). Clinically the patient may be breathless, but often is not. There may be a *sensu* of tightness across the chest, and the pulse rate and temperature may rise. On examination mediastinal shift to the side of the collapse may be detected, but cannot be relied upon. Percussion is impaired, and air entry absent, although

sometimes breath sounds are conducted across from the opposite lung. *Only radiological diagnosis is reliable* It must always be sought, and acted upon, even in the absence of symptoms.

Treatment consists of immediate bronchoscopy under local anaesthesia and thorough aspiration of the secretions in the blocked bronchus. It is quite futile to treat atelectasis by postural drainage. Often a tenacious mucous plug must be extracted with biopsy forceps. When all visible obstruction is removed, the patient is asked to cough while the sucker tube is left in place so that mucus in the more distal bronchi is driven within its reach. Postural drainage of the lobe is subsequently carried out. If re-expansion does not follow bronchoscopy and there is no other impediment, such as a fistula, to prevent it, bronchoscopic aspiration must be repeated at intervals until re-aeration is achieved (X-ray 12).

Bronchopleural fistula is much less common after lobectomy than after pneumonectomy, and when it does occur, is almost always in tuberculous subjects. As a rule it leads to collapse of the residual lobe as well as to infection of the pleural space, but if expansion of the remaining lobe is well established, and the lobe adherent to the chest wall, the resulting empyema is limited. If the space is that left by an upper lobectomy, drainage of it through the fistula itself may be sufficiently dependent, and the space can be very greatly reduced by an apical thoracoplasty so that ultimate disability is not great. If the fistula is in a lower lobe bronchus there is no hope of natural drainage, and even if the remaining lung largely fills the chest a so-called 'parabronchial pocket' will remain, and be the source of purulent, probably tuberculous, sputum. Such pockets call for exploration, resuture of the bronchus, the insertion of a muscle graft and dependent drainage. A basal dead space can sometimes effectively be reduced by the induction of a pneumoperitoneum.

After segmental resections an apical and basal drain are usually employed if the operation has been upon an upper lobe, but a single anterior tube is used if the lingular segment, or middle lobe, is removed, and a basal one only after excision of part of a lower lobe. Just as in the case of lobectomy their object is to get rid of all air or fluid in the pleural space impeding expansion of the lung, and continuous suction is always employed for at least 48 hours. Post-operative atelectasis and bronchopleural fistula are both uncommon, by far the most troublesome complication is persistent leakage of air from the raw surfaces of the lung adjacent to the removed segment. This is more likely to occur if the lung is emphysematous, fibrotic, or otherwise affected by previous disease. Such air leaks are

of two kinds—alveolar and bronchiolar. Alveolar leakage is inevitable whenever the lung parenchyma is cut across, and consists merely of seepage of fine air bubbles soon ceasing spontaneously in a healthy lung and promptly sealed by expansion against the chest wall. More troublesome leaks come from small bronchioles transgressing the division between segments. As has already been said, such leaks are searched for after resection and sewn up but some may escape notice and be the source of air leaks which do not heal spontaneously and quickly. Because the bronchioles involved are very peripheral infection does not follow leakage from them as it does from a bronchial fistula and the problem presented is wholly mechanical. They like alveolar leaks, are best sealed by rapid and complete re-expansion of the surrounding lung and its adherence to the chest wall. To ensure this the postoperative suction on both upper and lower tubes must be effective and continuous. If leakage is very severe, as it sometimes is from emphysematous lungs, care is taken to see that the volume of air extracted is the greater otherwise the air accumulates in the pleural cavity and compresses the lung. Should a tube become permanently blocked, or should lung re-expansion be irregular and air loculate in the pleura, a new tube is inserted into the air pocket by the use of an intercostal trocar and cannula so that it may be fully extracted. As most segmental resections are performed on upper lobes, and it is at the apex air tends to collect, it is helpful to nurse such patients lying on their operated sides in a slight head-down position for the first three or four postoperative days, the raw area of the upper lobe thus being brought early against the chest wall. Patients are practised in this position preoperatively. Should a leak prove considerable, and difficult to manage, and the lung has not expanded after three or four days, the chest should be reopened, the leak found and sewn up. Persistent leaks can also be dealt with and residual apical air pockets obliterated, by the removal of sections of two or three ribs overlying the troublesome area in order to bring the chest wall into contact with the lung, if the lung cannot be brought out to the chest wall. The performance of these very limited apical or lateral thoracoplasties seldom involves removal of the first rib.

Anaesthesia in chest surgery

A detailed account of anaesthesia is beyond the scope of this book. Briefly it may be said the less there is of it the better. The sooner a patient regains consciousness the sooner he can cough, expel his bronchial secretions and expand his lung. For this reason local

anaesthesia is always employed to infiltrate the operation field and to produce paravertebral and sometimes vagal block before the skin incision is made. The need for general anaesthesia is thus diminished. Anoxia must be avoided at all times. *Induction* is with intravenous Pentothal of which about 0.5 G. is usually required, but never more than 0.75 G. in the course of the operation. Pentothal is a dangerous drug when used in excess, and must especially be avoided in patients already suffering from anoxia. In the operation of pericardectomy for constrictive pericarditis it is better avoided altogether. *Relaxation* is achieved by the use of tubocurarine chloride, an initial dose of about 25 mg. being necessary in the average adult, followed at intervals by 3 mg. repeated as required. *Maintenance* consists solely of gas and oxygen except in very small infants when ether is preferred. The supply of oxygen is at all times generous, and carbon dioxide is absorbed within the circuit.

Two requirements are of prime importance in chest anaesthesia.

(1) Spill-over of pus, blood or secretions from the diseased to the healthy lung must be prevented.

(2) Respiration must be controlled throughout the operation by the anaesthetist.

Spill-over is avoided in one of two ways: either the patient is so placed on the operating table that drainage is gravitational, and can be aided by intratracheal suction; or some form of intrabronchial blocker is employed. Of these the best known and most efficient is that designed by Vernon Thompson. A long, fine, rubber catheter, made semi-rigid by a removable stilette, is passed down a bronchoscope under direct vision into the bronchus which it is desired to occlude. A small fabric-covered balloon at the end of the tube is then inflated with a few cc. of water so that it is held firmly in the bronchial lumen and effectively seals it, isolating the diseased lobe of the lung beyond. The stilette and bronchoscope are then withdrawn. A sucker passes through the centre of the balloon so that the lobe to be removed can be deflated, and no pressure of secretions is allowed to build up distally. A McGill tube is passed into the trachea, and the anaesthetic circuit effectively closed.

It is evident that a thoracotomy wound opening into the pleural cavity would result in a sucking pneumothorax (see p. 72), with gross paradoxical movement of the mediastinum were respiration not controlled and the anaesthetic circuit not closed. Such 'mediastinal flap' not only prevents adequate aeration of the opposite lung, but also filling of the heart. In thoracic surgery therefore the diaphragm and other muscles of respiration are wholly relaxed, and the

act of breathing lies literally in the hands of the anaesthetist who controls its rate and tension either by manually compressing a rubber bag in his closed circuit, or with the aid of a mechanical pulmo-flator. The mediastinum is thus kept steady and paradoxical movement prevented.

The use of an intrabronchial blocker has a further indirect advantage in patients undergoing pneumonectomy. If there is any sign of cyanosis when one lung is blocked off and 20 per cent. of oxygen is being delivered to the circuit it is clear the operation will not be well tolerated (even though ligation of the pulmonary vessels will subsequently direct unoxygenated blood to the aerating lung), and if possible a lobectomy should be performed instead.

THE CHEST WALL AND PLEURA

CONGENITAL ABNORMALITIES

CONGENITAL abnormalities of ribs are quite common but mostly unimportant. The first rib in particular is subject to variation, sometimes being fused with the second by a wide anterior plaque of bone; or unusually narrow, and accompanied by a cervical accessory rib which may give rise to symptoms of brachial neuritis. Complete absence of one or more ribs results in a soft lung hernia through the deficiency, moving paradoxically with respiration and coughing, and therefore a source of breathlessness and requiring repair by rib grafting, or with tantalum gauze, or plastic sheeting, to restore thoracic rigidity. Scoliosis in infancy causes chest deformity by rotating the bony cage, and the unilateral prominence of the costochondral junctions which results is sometimes mistaken for a tumour.

Minor defects of the sternum occasionally occur in combination with mediastinal dermoids; and gross ones are seen in infants with ectopia cordis, a condition incompatible with life.

A commoner congenital sternal deformity is *pectus excavatum*, or *funnel chest*. In this the manubrium is normal, but the lower part of the sternum, especially at the junction of xiphoid with gladiolus, is deeply depressed into the thorax, and may be almost in contact with the vertebral column. The lower costal cartilages are consequently acutely angulated. In the majority of patients the only resulting disability is a cosmetic one, but in some the displacement of the heart to the left causes palpitations; and in others there is collapse of the lower lobe of the left lung, with bronchiectasis. Under these circumstances, or if it is desired to improve the cosmetic appearance of the chest (sometimes an important consideration in women), surgical correction is indicated:

Through a midline incision the surface of the sternum is exposed and the insertions into it of the pectoralis muscles stripped off. The xiphoid process is excised, and the substernal ligament cut to mobilize the under-surface of the sternum from the pericardium and mediastinal contents. A wedge osteotomy is made transversely across the body of the sternum at the point where the depression begins, and

the deformed costal cartilages resected completely, until the lower portion of the sternum can be elevated into a normal position. The detached pectoralis insertions are finally sutured back to the sternal periosteum, maintaining it in the new position, and the wound closed. No other fixation, internal or external is required.

TRAUMA

The importance of injury to the chest wall depends upon the effect it has on the function of the underlying lung and the treatment of such injuries always has for its object (in addition to the usual measures to combat shock and replace blood lost) the prompt restoration of normal lung function. In some cases this will involve closing open wounds, or preventing paradoxical movement by restoring rigidity to some part of the thorax in some removing blood or air from the pleural cavity to permit pulmonary re-expansion, in some, performing bronchoscopy and sucking out oedema fluid, or secretions, in those unable to cough and so to maintain a clear air way and in others merely taking measures so to relieve pain that breathing and coughing are effective. In all an early chest X ray is imperative, and more informative than any physical examination, though percussion is useful in rapidly establishing whether air or fluid are in the pleural space. Some degree of upper abdominal rigidity is common after chest trauma and does not necessarily mean that a wound is a thoraco-abdominal one or that an abdominal viscus is injured though the frequency with which splenic rupture is combined with a left-sided crush injury of the chest should be borne in mind.

Non-penetrating injuries

These greatly predominate in peace-time and, apart from simple rib fractures, traffic accidents are their most prolific source, the commonest of all being the impaction of a driver against his steering wheel. To them in war time is added blast injury of the lungs caused by bomb or shell explosions. Accidents of this kind, which involve crushing or compression, may fracture ribs or the sternum, but can severely injure the underlying lungs without doing so.

A fracture of the bony cage may call purely for local treatment, or result as well in a haemothorax with which it is far more important to deal. Whereas if the chest is struck, or exposed to blast while the glottis is closed, no bones may be broken, but the sudden intense positive pressure generated in the lung is likely to rupture alveoli, letting air escape into the tissues or into the pleural space, as well

as causing bleeding, so that haemoptysis, pulmonary contusion, surgical emphysema, pneumothorax or haemopneumothorax may all follow.

1. *Fractured ribs*

If the fracture is a simple one, and due to antero-posterior compression, it usually occurs at the outer convexity of the rib in the axilla, and is undisplaced, so that lung injury is rare. Shearing forces may cause multiple breaks through the rib necks, and these sometimes comminute and tear the lung to produce a pneumo- or haemopneumothorax. Severe direct blows may fracture ribs at both ends, depressing a portion of the bony cage and leaving it free to move paradoxically with respiration.

It is only in the latter instance, which is rare, that fractured ribs should be 'strapped up', for it is then necessary to control and minimize paradoxical movement. In other cases it is highly undesirable to immobilize the injured chest, and immobilization is the whole object of strapping. Broken ribs are effectively splinted by their neighbours, and the effect of strapping a chest is merely to provide so much additional discomfort that the fracture pain is forgotten. A more sensible way of dealing with pain is to block the affected intercostal nerve or nerves with a long-lasting oily solution of procaine. This, by abolishing the pain on coughing and deep breathing, makes the chest more mobile, not less.

2. *Fractured sternum*

Fracture of the sternum is always the result of direct crush injuries, and is usually undisplaced, calling for no special treatment beyond direct injection of a local anaesthetic. If the bone, however, is unstable, and paradoxical movement is present, this must either be controlled by strapping the chest, or by fixing the two portions of the sternum with a Kirschner wire or by direct wire suture.

3. *Haemothorax*

Bleeding into the pleural cavity after injury, whether penetrating or not, is the commonest and most important complication of thoracic trauma. Bleeding of this kind is invariably from the systemic vessels of the chest wall itself, usually the intercostals, and not from the lung in whose vessels the pressure is much lower, unless there has been direct injury to a major pulmonary artery or vein, in which case survival is unlikely. It is perfectly possible for several litres of blood to accumulate, compressing the lung, displacing the mediastinum,

and exsanguinating the patient. Under these circumstances there will be increasing breathlessness, cyanosis and shock, and prompt intervention is life saving. On two occasions during the last war I was confronted with this syndrome in places remote from facilities for blood transfusion. Both were non penetrating crush injuries with purely superficial abrasions and both patients were bleeding to death into their chests, and being rapidly suffocated by mediastinal displacement as they did so. The blood in the chest was aspirated through one needle connected with a syringe and two-way tap thereby relieving the respiratory distress, and pumped straight into an arm vein through a second needle thereby transfusing the patient with his own blood. In both instances improvement was dramatic and immediate.

Such cases are extreme but even a small haemothorax must be completely removed as soon as possible. At best it prevents full lung expansion, and daily deposits fibrin whose layers slowly thicken upon both lung and chest wall, immobilizing them. The blood itself acts as an irritant to the pleura and causes it to pour out an effusion, which dilutes the blood but increases the fluid, impeding aeration of the lung. Worse still, and contrary to popular belief blood in the pleural cavity *does* clot (in about 10 per cent. of traumatic haemothoraces) especially if tissue damage is considerable and cannot then be aspirated at all, so that the whole mass consolidates and becomes organized to freeze both lung and chest. Finally large blood clots form perfect culture media, and should secondary infection occur empyema follows. The risk is of course much greater in the case of a penetrating wound.

The best treatment of a haemothorax is immediate and complete aspiration, and this should not be delayed even for blood replacement. A haemoglobin estimation of the fluid removed to determine how much of it actually is blood, and how much secondary effusion, is valuable. The notion that early aspiration of blood from the chest is dangerous because it stimulates fresh bleeding is merely one of the many old wives tales that bedevil medicine: the sooner blood is removed the less likely it is to clot and the sooner will the lung be able to re-expand. If on the other hand, bleeding from the chest wall continues, as evinced by the patient's condition, pulse rate and blood pressure by a high haemoglobin content of the aspirate, and by the continued appearance of bright blood in it, there is only one sensible measure, and that is prompt thoracotomy to secure the bleeding point, and to clear out the clot.

Should partial clotting take place before aspiration is complete,

so that pockets of serum become loculated, the effect of the enzymes streptokinase and streptodornase should be tried, 40,000 (Christiansen) units of the first and 10,000 of the second being injected into the haemothorax on three alternate days with aspiration on the intervening days. The reactionary fever is controlled with aspirin. Although these enzymes will often make it possible to remove the semi-fluid gel, they do not dissolve away the fibrin already deposited on both layers of the pleura.

In most cases of clotted or infected haemothorax formal thoracotomy with evacuation of all the pleural contents, and decortication of the lung and the chest wall, is necessary. In early cases this can be done manually and by swab dissection, but in later ones, in which the fibrin has become organized, deliberate decortication will have to be carried out in the same way as for a chronic empyema. At the end of the operation the lung is re-expanded, two drains connected with suction are left in the chest, and the intercostal wound closed. Postoperative breathing exercises are almost as important as the operation itself.

4 *Blast*

When an animal such as man is subjected to explosive blast his flexible ribs are driven in against the lung, bruising it and causing local oedema and extravasation of blood which may lead to pulmonary collapse. In addition, should the blow occur when the glottis is closed alveoli are ruptured by the sudden positive pressure, allowing air to escape into the tissues as surgical emphysema, or into the pleural space to produce a pneumothorax. The ensuing anoxia and shock are treated by rest and oxygen; impaired coughing is assisted by bronchoscopy, with intrabronchial suction of blood and oedema fluid to help re-expand collapsed segments; and blood or air in the pleural cavity is removed by aspiration.

5. *Mediastinal (or interstitial) emphysema*

After injuries such as the foregoing, or crushing accidents, air may be forced out of ruptured alveoli into the interstitial tissues of the lung to track proximally into the mediastinum, and present as a soft tender crepitant swelling at the base of the neck. Sometimes the face, too, becomes swollen. Usually such air, though a cause of great discomfort, is reabsorbed within 48 hours, but should it continue to increase, spreading out in other tissue planes and even reaching the finger-tips and scrotum, a wound of the lung or of a bronchus is suggested, which calls for exploration and repair. Several instances

are on record of a main bronchus shorn off by a violent, non-penetrating blow on the thorax, and successfully restored by immediate suture.

6 *Pneumothorax and haemopneumothorax*

These may complicate non penetrating injuries for the same reason as the above, or because of direct lung puncture by a comminuted rib fracture. They are, however an almost invariable accompaniment of penetrating chest wounds, and so will be considered in the next section.

Penetrating injuries

These greatly predominate in war, and account for 10 per cent. of the wounded surviving beyond an advanced dressing post. In peace-time they are largely confined to stabbing affrays. Air as well as blood is likely to be found in the pleural cavity, and the risk of infection by organisms carried in from without is of course much greater. This risk is much less with high velocity bullets than with low velocity high-explosive fragments, which are the commonest missiles of modern war, and in addition, are jagged and apt to carry into the chest contaminated clothing particles. It is surprising how often stab and bayonet wounds remain sterile simply because they do not do this and are sharply incised.

1 *Pneumothorax*

This is the simplest complication of a stab wound in the chest, and in most cases calls simply for the removal of air by an artificial pneumothorax apparatus, such as the easily portable Maxwell box, as the lung wound rapidly seals itself off (X ray 15). It is, however, frequently accompanied by bleeding from the chest wall or from the lung, so that in the great majority of penetrating chest wounds, especially in war, the condition is one of haemopneumothorax. (For *Spontaneous Pneumothorax* see the chapter on 'Cysts of the Lung')

2 *Haemopneumothorax*

Here, assuming bleeding has stopped and thoracotomy is not required, both blood and air must be withdrawn as soon, and as completely as possible in the same way as that already described for haemothorax (X ray 13).

3 *Tension pneumothorax*

Should the wound in the lung prove valvular a positive air pressure may build up in the pneumothorax so that the heart and

mediastinum are progressively displaced to the opposite side, and the patient becomes increasingly breathless and cyanotic (X-ray 15). The condition is instantly relieved in emergency by the insertion of a needle—any needle—into the pleural cavity as a safety-valve, followed as soon as possible by the removal of all the air. If the valve mechanism continues to function, the tension element will recur, and must then be dealt with either by an indwelling needle of the Foster-Carter type, or by an intercostal catheter, connected to water-sealed suction (Fig 22) until the lung re-expands and seals off the leak.

4. *Sucking pneumothorax*

If the chest wall wound remains open, or a section of the wall has been blown away leaving a deficiency, air can pass freely in and out with every breath—an extreme degree of paradoxical respiration. The underlying lung is collapsed, and the function of the opposite one is almost as severely deranged, for air from it passes just as readily into the wounded side as through the glottis, both being at an equal pressure. Carbon dioxide builds up, oxygen falls, and very severe dyspnoea and cyanosis precede death if nothing is done. First aid consists in closing the defect at once with a layer of *tulle gras*, or waterproof silk, held firmly in place by a bulky dressing until such time as the wound can be cleaned, excised and resutured; but, failing that, a field dressing or even a pad of unsterile clothing strapped over it will be life-saving.

5. *Surgical emphysema*

Some air escapes into the tissue planes of the chest wall after most wounds of the chest, surgical or traumatic, but it is generally slight in extent and rapidly reabsorbed. It differs in no respect from that already described under *mediastinal emphysema*, and should it increase also demands intrathoracic exploration, as a penetrating wound of a bronchus or the trachea may be present.

6. *Thoraco-abdominal wounds*

The trajectory of a bullet which has no exit wound is speculative, and many war wounds involved both pleural and peritoneal cavities. On the left side the spleen is particularly vulnerable to both penetrating and non-penetrating injuries, and rupture of the diaphragm on that side leads to herniation of stomach or bowel into the thorax (X-ray 109). On the right the liver forms an effective barrier, but bile may drain into the pleura and be aspirated from it. The surgical

approach to such injuries should be through the chest in the first instance so that vital structures can be dealt with, and a wide exposure obtained of the track of the missile. Splenectomy or repair of the stomach can very conveniently be carried out through the left diaphragm. For more extensive injuries the thoracotomy incision is continued down on to the abdominal wall to become a laparotomy.

INFLAMMATORY LESIONS

1. *Empyema necessitatis*

An undrained empyema is eventually likely to point through an intercostal space, usually on the front of the chest, as a fluctuant swelling which gives an impulse on coughing, and whose contents can be expressed back into the pleural cavity. If the empyema is a pyogenic one the skin over it may be reddened and tender, but if tuberculous (and nowadays this is the commoner of the two) there may be neither heat nor pain. The recognition of an empyema necessitatis of this kind is an urgent indication for rib resection and drainage at the site of election—that is the most dependent point of the empyema as determined by radiography after the injection of 10 cc. of Lipiodol—and *not* aspiration still less summary incision of the fluctuant mass, for the latter is very rarely at the site of election and needle puncture of it will probably lead to a persistently discharging sinus.

2. *Tuberculous abscess*

Tuberculous lymphatic glands situated at the anterior ends of the intercostal spaces often break down to form a small, deeply situated, fixed and tense cold abscess from which fluctuation can nevertheless with some difficulty be elicited. It is important to distinguish this type of abscess from a tuberculous empyema necessitatis whose treatment is radically different. Apart from its firmness, and the absence of radiographic evidence of gross pleural disease the abscess gives no impulse on coughing, and the pus in it cannot be expressed back into the chest. It may however be aspirated, and will generally prove to be sterile. The best treatment is complete excision of the breaking-down gland with primary suture of the wound. Sometimes secondary chondritis of costal cartilages is present and requires radical excision and sometimes sinuses track deeply into the chest wall from the presenting abscess. All these must be pursued, and either thoroughly curetted, or excised with any involved cartilage.

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Streptomycin is dusted into the wound before closure as well as being given by injection.

3. *Osteomyelitis of ribs*

This is now rare, but should it occur is likely to be associated with an inadequately drained empyema. Rib sequestra are occasionally found in the chest wall sinuses, and are a contributory cause of continued discharge from them. They call for exploration, excision and definitive drainage.

4. *Tietz's disease*

This is a non-specific, non-suppurative condition causing painful swelling of one costo-chondral junction. Seldom encountered in civil practice, it is remarkably frequent among young military recruits and often leads to unnecessary operations. Pack drill has been held responsible. It runs a long course, the swelling altering in size but never breaking down. Histologically there is some periarticular swelling and fibrotic change in the cartilage.

5. *Typhoid osteomyelitis* and *gummas* are now rare, but the former may appear long after the original typhoid infection.

NEW GROWTHS

Neoplasms of the chest wall are divided into the superficial and the deep. The former are mainly innocent lesions such as cysts, naevi and lipomas, which originate in the skin and subcutaneous tissues and are therefore mobile, the latter involve ribs and intercostal muscles, must all be regarded either as malignant or potentially so, and are fixed. The superficial group differs in no respect from similar lesions elsewhere and calls for no further comment; but tumours involving ribs have certain special features.

Chest wall tumours often give rise to segmental pain long before swelling is apparent, for the neoplasm is able to expand almost indefinitely into the pleural cavity instead of appearing on the surface. The pain is boring or pleuritic in character if local, but when it is referred to the abdominal wall along the segmental nerves it may lead to mistaken diagnoses of gall-bladder or other intra-abdominal disease, and it has been estimated that on the average six months elapse between the onset of such pain and X-rays being taken of the affected part, and two of them are spent in treating 'neuritis'. Secondary invasion of the chest wall by tumours arising in the lung, and metastases in ribs, are almost always painful, and are much

commoner than primary tumours which, though they do cause pain often attain considerable size before doing so. Pathological fracture may first draw attention to a neoplasm, and must be distinguished from idiopathic 'cough fractures' usually seen in the first, second, or eighth ribs, and attributed to stresses resulting from coughing, in which the surrounding bone is quite normal.

1 *Secondary invasion by bronchial carcinoma*

This is incomparably the most common source of persistent chest wall pain, rib erosion and non-fluctuant swelling in men over 40 and should first be considered in all cases. Unless it involves the brachial plexus or the vertebral bodies, every effort should be made to remove such a growth, the affected segment of the chest being radically excised together with the lung. This is of the greatest value for the relief of the otherwise intractable pain, quite apart from the fact that many such growths are otherwise readily operable. The continual toothache like pain which accompanies chest wall involvement by inoperable neoplasms can sometimes be relieved by *intercostal neurectomy*.

Under local or general anaesthesia a vertical incision is made parallel to and about three fingers breadth from the vertebral spines and deepened to expose the transverse processes, and extreme posterior ends of the affected intercostal spaces. The intercostal muscle fibres are cut and each intercostal nerve is found at this point lying just superficial to the pleura, and about midway between the ribs, the intercostal artery crossing it to reach the subcostal groove of the rib above. The nerve is picked up with a hook, care being taken not to tear the pleura, and a section of a few centimetres cut out of it. This is repeated in each space until all the involved nerves are divided.

Most so-called endotheliomas of the pleura are in fact anaplastic peripheral bronchial carcinomas which invade the pleura early and become disseminated over it. True endotheliomas are excessively rare.

2 *Metastases*

Metastases account for about 40 per cent. of all chest wall tumours and are often secondary to carcinomas of the lung, thyroid, kidney and breast, preceding in some instances recognition of their primary source, and being mistaken for intrinsic rib tumours. When very vascular they may present as pulsating masses and at first sight resemble an aortic aneurysm which can similarly produce a pulsatile

thoracic swelling, usually to the right of the sternum, but which is in fact not invariably pulsatile.

3. *Fibrosarcoma*

Fibrosarcoma of the chest wall is fortunately not common, but distressing when it does occur in that it is virtually untreatable. Pain is not conspicuous at first and the growth is therefore able to infiltrate extensively before it gives rise to much swelling, so that excision holds out no hope of cure. The tumour is not sensitive to deep X-rays. Intercostal neurectomy affords palliation.

4. *Primary tumours of ribs and sternum*

In a review of 48 such tumours Blades and Paul found 28 benign, of which at least 7 were potentially malignant, and 20 (or 41 per cent) frankly so. Such growths of the thoracic cage comprise about 10 per cent of all primary malignant bone neoplasms, the ribs being involved in 80 per cent, the sternum in 20 per cent. Osteogenic sarcoma and osteoclastoma of these bones, in contrast to those elsewhere, are, however, exceedingly rare, there being only 9 examples of the latter recorded.

Much the commonest primary tumours are:

(a) *Chondromas and chondrosarcomas* which between them account for half the total. 70 per cent of them grow anteriorly near the costochondral junction (X-ray 16), and the manubrium is the most commonly affected portion of the sternum. As their name suggests, both are essentially growths of cartilage, bone formation or mucoid degeneration in them being merely secondary features, but leading to much confused nomenclature such as 'osteochondroma', 'osteochondrosarcoma' or 'chondromyxosarcoma'. Indeed it might be better were no distinction to be made between innocent 'chondromas' and 'chondrosarcomas'; for all the former are potentially malignant, and it may be virtually impossible to draw a line between them even on histological grounds.

Most cartilagenous tumours are at first hard, nodular and fixed; but as they get larger, or recur, they develop softer or even fluctuant areas of mucoid degeneration. Radiologically destroyed bone is visible, with an expanded and thinned-out cortex; and flecks of irregular calcification throughout the mass are characteristic. As a preoperative diagnosis cannot be made with any certainty, much less a distinction between innocence and malignancy, all should be radically extirpated along with the adjoining muscles and pleura. Defects in the chest wall which permit paradoxical movement are

repaired, if they are substantial either by interposing rib grafts from above and below, by rigid prostheses of plastic sheeting, or tantalum gauze or, if small, by transposing periosteum across them so that new bone is formed. Most of the tumours seem to be encapsulated, and even malignant ones can be shelled out with deceptive ease. The advancing edge of the growth remains discrete, first displacing and distorting adjacent soft tissues, then invading them, and local recurrence increasingly prompt and increasingly malignant, follows inadequate removal.

A farm labourer aged 32 first had a histologically proven simple chondroma removed from the anterior end of his third rib twelve years prior to the reappearance of a swelling in the same site. This too was excised and again said to be an innocent chondroma. Eighteen months later a partly firm, partly fluctuant mass reappeared. It was resected together with several adjacent ribs and part of the sternum, its ramifications calling for wide thoracotomy. It showed much mucoid degeneration, and in spite of the fact that mitoses were hard to find, was undoubtedly sarcomatous.

This story is typical and mitoses are always exceptional, the presence of atypical nuclei or giant cartilage cells indicating malignancy. Enchondral bone formation suggests this to be low whereas if mitoses are seen it is likely to be high. Some portions of a tumour may seem innocent, others invasive. Metastases are unusual, appearing only late in the disease, and generally being blood borne. The tumours, like most others growing from the chest wall (with the exception of Ewing's sarcoma) are quite irresponsive to deep X rays.

(b) *Fibrous dysplasia* is the next commonest rib tumour and is usually seen in young adult males. It is often symptomless, and discovery follows trauma. Radiologically the rib shows fusiform expansion, with a narrowed cortex and semi translucent centre. Histologically the medulla is replaced by masses of young fibrous tissue in whorls and trabeculae with patches of bony metaplasia. Malignant change does not occur and local excision of the affected rib is sufficient.

(c) *Rarer tumours* are

Single myelomas which may closely resemble the foregoing radiologically and are like myelomas elsewhere, except that Bence-Jones protein is not usually present in the urine. The plasma proteins however may be raised. There is some tendency to recurrence but usually local removal suffices.

Plasmacytomas and *eosinophilic granuloma* are occasionally seen mostly in male children and adolescents, with symptoms of local

tenderness or a pathological fracture. They are accompanied by eosinophilia, pyrexia and loss of weight, and the prognosis following excision is good

Ewing's tumour of ribs is also rare, but when it occurs is found in patients during the second decade of life, and should be treated by deep X-ray therapy, to which it responds.

EMPHYEMA

EMPHYEMA is probably the worst treated of all common disorders of the chest. This is largely because the overriding importance of the physiological factors concerned is not kept clearly in mind, the element of speed in treatment is neglected, and certain simple but vital technical steps are ignored. Failure in treatment, at the very best, leads to serious and permanent impairment of pulmonary function at the worst it costs the patient his life. Between these two extremes lies chronic illness sometimes dragging on for many years.

The chef of a great London hotel was the patient of a colleague of mine the man had served with the French artillery in 1914 and been shot through the upper part of his left chest. An empyema resulted which was drained at a base hospital, and according to the bad practice of those days (and sometimes of these) the tube was removed when no more pus came out of it. The wound healed, and he was discharged but he did not feel well, and occasionally had a raised temperature. Invalided from the army he continued to have bouts of malaise and fever. After an abscess appeared on the front of his chest, and was incised, he felt better and took a post as chef. In the ensuing years he had several illnesses diagnosed as pneumonia and during one of them coughed up some blood, and then a considerable quantity of pus. He was told his wound had 'weakened' his lung, and an abscess had formed. Again he recovered, and again redness and swelling appeared below the scars of his old injury, and pus trickled from a sinus in their midst. After this had been dilated with forceps, drainage occurred, the sinus healed for a time, but reappeared lower down his chest. Years passed, and although he prospered in his profession and took many Riviera holidays on medical advice, his health remained bad, he was subject to recurrent fevers and wore a bulky dressing to absorb the pus trickling from sinuses which repeatedly broke down on his chest. In addition, he underwent many operations and expended a large sum in medical and surgical fees. At the end of forty years, and after a second World War he had the good fortune to consult my colleague, who drained a residual empyema below the patient's left costal margin, and brought the sorry saga to an end. Such are the consequences of taking the drainage tube out of an empyema too soon.

The diagnosis of 'empyema' is inadequate. It is never a primary condition, but always secondary to injury or disease elsewhere. Infection may come from the lung, from the oesophagus, from below the diaphragm, or through the chest wall. As the primary lesion may be of much graver import than the empyema which is a symptom of it, it is always necessary to answer the question 'secondary to what?'

Aetiology

The three commonest causes of empyema are *lobar pneumonia*, *carcinoma of the lung* and *pulmonary tuberculosis*. As a tuberculous empyema is always associated with a tuberculous lesion in the underlying lung, it poses special problems of management and is discussed separately in the chapter on the surgery of tuberculosis, although the fundamental principles underlying treatment are essentially the same.

So far as inflammatory lesions in the lung are concerned, the widespread (and often promiscuous) use of antibiotics, although it has diminished the incidence of empyema after *lobar pneumonia*, has by no means abolished it. Indeed, because symptoms are suppressed, there is a greater risk of an empyema passing undiagnosed into a chronic state when its ultimate treatment is much less likely to be satisfactory. Infection from a pneumonic lung may extend to its visceral surface causing first pleurisy, and then an effusion which rapidly turns to pus. The same thing may complicate a *lung abscess*, if the abscess is prevented from draining spontaneously into the bronchial tree; and follows the rupture into the pleural cavity of any *infected cyst*, whether it be bronchogenic, an hydatid, or a mediastinal dermoid. The thin walled cyst-abscesses typical of *staphylococcal pneumonia* in infancy often cause empyema in this fashion so early in the disease that the pleural infection is mistaken for a primary condition, the pneumonia having already cleared from the lung before the pleural pus is diagnosed. *Chronic bronchiectasis* is characterized by periodic acute exacerbations during which pleurisy may precede an empyema; but because previous pleuritic attacks have already caused pleural adhesions, such collections of pus are likely to be limited and loculated; for an empyema may involve the whole or only a circumscribed part of the pleural cavity. Of all pulmonary lesions leading to empyema the one that is most essential to diagnose correctly is *bronchial obstruction* when a bronchus is blocked by growth, by a stricture, or by an inhaled foreign body, mucus is dammed behind the blockage and becomes infected; and this infection is likely to extend to the pleura unless the obstruction is relieved (X-rays 45, 46). Far and away the commonest cause of such obstruc-

tion is bronchial carcinoma it follows that *any empyema occurring in a man of cancer age must be assumed to be due to a growth until that possibility has been eliminated*. Bronchoscopy is imperative in every case and there can be no more lamentable error than to drain an empyema and leave behind a neoplasm. Nor does the existence of an empyema by any means indicate a growth to be inoperable unless a necrotic tumour is actually breaking down into the pleural cavity. In children bronchial obstruction calls equally for bronchoscopy for it may be due to a non radio-opaque inhaled *foreign body*, or in young adults (especially women), to an *adenoma* (X ray 45)

Infection may reach the mediastinal aspect of the pleura from a *perforation of the oesophagus*, of which the commonest cause is certainly the unskillful passage of an oesophagoscope. Other foreign bodies cause less trouble, but if they are sharp sometimes penetrate the wall. Even so damage more often follows attempts to remove them just as rupture at the site of an oesophageal carcinoma usually accompanies unwise instrumentation such as dilatation of a malignant stricture, and not erosion by the growth itself. Rarely too a peptic ulcer at the lower end of the oesophagus perforates and spontaneous rupture occasionally occurs in debilitated alcoholics during a bout of vomiting. The chief hazard of oesophagectomy is leakage from the anastomosis and empyema.

Subphrenic abscess is the commonest source of pleural infection from below the diaphragm. Because these abscesses tend to be diagnosed late, or not at all, and are tightly enclosed by the liver and its ligaments, a high proportion are accompanied first by an effusion above the diaphragm, and later by pus. Infection does not occur in the reverse direction, because it is easier for an empyema to rupture into the lung, or to extend elsewhere in the pleural cavity, than to pass the barrier of the diaphragm.

Penetrating wounds of the chest cause empyema if infection is carried into the pleural cavity but this happens surprisingly seldom in peace time. Most stabs result in haemopneumothorax, as do high velocity bullets (with their clean shape) in war. High-explosive fragments on the other hand cause ragged wounds, and because of their low velocity carry infected clothing into the chest with them.

Finally empyema is the inevitable consequence of *broncho-pleural fistula*. This postoperative disaster used to occur in some 60 per cent of all resections of the lung, was frequently fatal and constituted the chief factor limiting the progress of thoracic surgery. With the advent of antibiotics such as penicillin and streptomycin, and of improved techniques it is now a comparatively rare complication of surgery,

involving less than 1 per cent. of pneumonectomy patients, and fewer still of those undergoing lobectomy. A good deal of the improvement in survival figures following resection for carcinoma of the lung in recent years has been due to the virtual elimination of this accident

Diagnosis

An acute empyema supervening upon lobar pneumonia is recognized by the following changes.

Already ill, the patient becomes more toxic in appearance; and fever, hitherto sustained (unless masked by antibiotics), alters to the remittent type associated with an undrained abscess. As fluid accumulates, the patient becomes more breathless as the lung is compressed, and the heart and mediastinum are displaced towards the opposite side. Physical signs correspondingly alter, movement becoming less, and the percussion note, already impaired by the consolidated lung, is further dulled by the intervening pus. The bronchial breath sounds previously audible disappear. Vocal fremitus, increased in pneumonia, is absent. The white cell count rises above 20,000; and radiography of the chest shows a new and additional opacity rising like a meniscus in the axilla if the pleura is free, but encysted, and related to the diseased part of the lung, if the accumulation is limited by adhesions. Sometimes pus may be confined in one or other of the lung fissures, or between the lung and the mediastinum or the diaphragm, so that it is not directly related to the chest wall and aspiration is rendered difficult. Such abscesses are uncommon. These signs and symptoms, and especially fever and evidence of toxicity, may be diminished or completely masked by antibiotics, so that the empyema escapes diagnosis, to make its presence felt much later. This is more likely to happen if it is confined by adhesions to one small part of the pleural cavity.

Treatment

1. Aspiration

The lines on which an empyema is treated depend on how long it has been present, the thickness of the pus it contains, the degree of mobility retained by the lung, and on whether or not the latter is still diseased. All methods of treatment, however, have a single object which must constantly be kept in view: *to restore as quickly as possible, and as completely as possible, the normal function of respiration.* Providing the lung itself is not diseased, this function depends upon two equally important factors:

- (1) the full expansion of the lung,
- (2) the maximum mobility of the chest wall, which includes the diaphragm.

With each day pus remains within the pleura a new layer of fibrin is deposited upon the lung and the parietes, corseting them both with increasing rigidity and making their remobilization more difficult. Prompt diagnosis and speedy and effective treatment are therefore essential. It is futile to do all the right things in so tardy a fashion that an acute empyema develops into a chronic one futile to secure re-expansion of the lung in a thoracic cage so frozen by fibrosis that the pulmonary pump no longer works or to devote much labour to breathing exercises and to the promotion of mobility if all the time the lung is unable fully to expand because of some mechanical impediment.

Once the diagnosis has been made the position of the empyema is determined as accurately as possible by percussion and by good postero-anterior and lateral X rays. These investigations do not indicate the lower limit of pus if as is most common the basal part of the thorax is involved, for dullness on percussion and radiographic opacity are then continuous with those of the liver below (X rays 17, 19 23). This is important, for the next step in confirmation is aspiration, and the commonest cause of failure in this is an attempt to insert the needle into the lowest part of the abscess. As we have just seen, it is probable that there is no real evidence where this is and in any case the lowest is the *least* satisfactory point for aspiration because sediment, fibrin and slough gravitate to this point and render aspiration there most difficult or even quite impossible. A spot should be chosen instead at the point of maximum dullness a short distance below the upper pole of the empyema. The aspirating needle can then be dipped down into the pus as far as is necessary. It is essential at this early stage when the pleural layers are not yet adherent, that no air be admitted into the empyema, either by accident or intent, lest it cause the visceral and parietal layers of the pleura to separate, and so convert a localized collection of pus into a total empyema (X ray 19). This is in every way analogous to rupturing an appendix abscess and causing general peritonitis besides admitting air as a further impediment to the re-expansion of the lung and is on all counts completely indefensible. Yet I seldom see a collection of fluid in the chest, infected or otherwise, into which air has not been admitted usually because unsuitable equipment has been employed, but quite often deliberately introduced in the erroneous

belief that 'the lung should not be expanded too quickly' or that 'the fluid level makes it easier to aspirate'.

The simple apparatus needed to perform a pleural aspiration consists of a 20-cc. syringe with a two-way tap and needles of bores varying with the thickness of the pus (Fig. 23). The two-way tap is

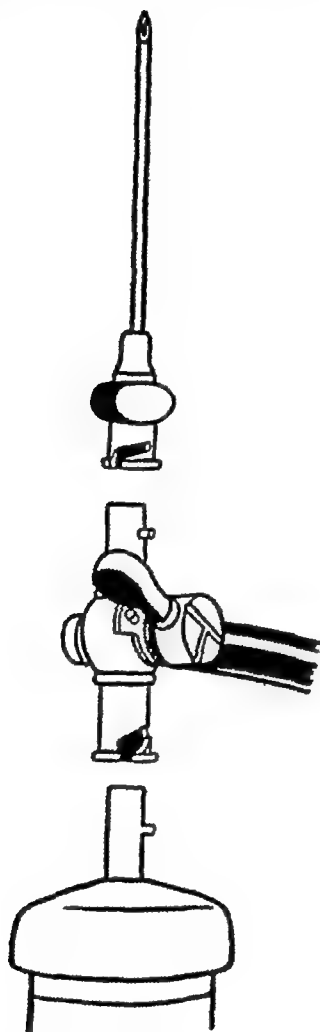


FIG 23

A Martin syringe with bayonet fittings and two-way tap.

connected to the syringe, and the needle to the tap, by either a bayonet or a screw fitting so that at no time can they become disconnected and allow air to be sucked into the chest. A record fitting is wholly unsuitable and unsafe and should never be employed. One of the most efficient types of aspirator is the Martin syringe with its two-way connection (Fig 23).

Before the pleural tap is performed it is important that both patient and surgeon should be made as comfortable as possible for it may be necessary that both remain in one position for some time. If the aspiration is to be made at the back it is best for the patient to sit forwards, leaning his head and arms upon a pillow placed on the bed table.

The position of the pus is now estimated in the patient's chest by measuring, and by counting ribs in comparison with the X ray findings. This is confirmed by percussion as being the area of maximum dullness. A point is chosen at the lower border of a suitable intercostal space, where there is no risk of damage to the intercostal vessels and nerve running along the inferior border of each rib and, after the surrounding skin has been painted with iodine or acriflavine in spirit, and sterile towels arranged, a skin bleb is raised with 0.5 per cent. procaine. Sufficient local anaesthetic is used to ensure that the whole procedure is quite painless and the infiltration is carried down to the parietal pleura, beyond which a trickle of pus appears in the anaesthetic syringe thus providing a useful measure of the depth of chest wall to be penetrated. The aspirating needle firmly engaged to the two-way syringe, is advanced through the chest wall until pus is located. As soon as some is withdrawn a pair of rubber tipped artery forceps is lightly clipped on the needle flush with the skin, as a marker and to avoid the tendency of the needle point to see-saw backwards into the chest muscles or forwards to prick the lung, and so possibly cause a pneumothorax. The first pus withdrawn is taken in a sterile test tube for culture and estimation of antibiotic sensitivity. A further tube-full is kept in a rack in the ward for comparison with the pus obtained at subsequent aspirations and finally *all* the remaining purulent fluid that can be aspirated is removed. It is quite wrong to take only an arbitrary quantity or to imagine that the patient will be distressed if much is removed at one time. The object of the operation is to secure the complete re-expansion of the lung and its adherence to the chest wall, if possible at the first aspiration (X rays 17-18). This becomes more difficult each day infected fluid remains in the chest. Every drop of pus should therefore be evacuated as soon as may be and, providing the procedure is carried out painlessly and efficiently there are no contra indications to this rule. The larger the effusion present the greater is the compression of the patient's lung and the displacement of the mediastinum, and the sooner they return to normal the better. Before the needle is finally withdrawn 1 000 000 units of penicillin are instilled into the infected space for no matter how high a blood

level of penicillin is attained by intramuscular injection an adequate amount does not reach such walled-off cavities unless it is directly injected into them. If the organisms subsequently prove penicillin-resistant, some more suitable antibiotic is substituted at the next aspiration. For success on the first occasion, although the ideal of treatment, is not often achieved, and aspiration must be repeated as often as fluid accumulates, perhaps daily, or every second day, but always with the aim of securing the complete re-expansion of the lung and consequently the obliteration of the space.

2. *The use of enzymes*

With the most conscientious efforts, however, or in those empyemas recognized less early, it may be found that aspiration becomes more and more difficult, even with a wide-bored needle, as the pus thickens. Pneumococcal pus in particular contains much clot; but streptococci secrete lysins which dissolve the fibrin and keep streptococcal pus fluid. These enzymes can be employed therapeutically to restore fluidity and so to prolong the time during which aspiration can be carried out and surgical drainage avoided. The empyema cavity is injected with 40,000 (Christiansen) units of streptokinase and 10,000 units of streptodornase on three alternate days, aspiration being carried out on each of the intervening days. As the fibrinolysins do their work the clots are broken up and the pus become more fluid, but these enzymes do not dissolve fibrin layers that have already been deposited on the pleural surfaces. The longer the time that has elapsed, therefore, the less effective will be their use. Some fever follows their injection, and is controlled by antipyretics.

3 *Surgical drainage*

Open surgical drainage becomes necessary if, in spite of these measures, obliteration is not achieved, or if the empyema is already in a chronic state when it is first diagnosed. When such a stage is reached there is no risk in opening the cavity because the pleural layers are by that time securely adherent. If 10 cc. of Lipiodol are injected into the empyema they sink to its bottom providing a clear marker of the most dependent point for drainage. After postero-anterior and lateral X-rays have been taken, the ribs are counted in order to select the most suitable one for resection. The operation is usually performed under local anaesthesia with the patient sitting upright on a stool and leaning forward with the arms resting on the operating table which is adjusted to a comfortable height. This position is preferred lest a bronchial fistula be exposed when slough is

removed, and pus flood into the lungs, as might happen with the patient lying on the operating table.

A vertical incision is made over the portion of rib which has been selected for excision, for should it prove necessary to insert the tube lower down, such an incision can conveniently be extended and the drainage tube sits more comfortably in a vertical incision than in one in the line of the rib. Enough of the chosen rib (usually about 4 inches) is resected subperiosteally (see Fig. 12) to provide adequate access for a thorough inspection and toilet of the cavity which is entered by incising the rib bed. All the pus is sucked out, as well as the sedimented fibrin and debris on the floor. A retractor and malleable light are then inserted and the curtains of slough which are seen hanging from the walls of the empyema are stripped away with sponge-holding forceps. When a complete toilet has been performed the walls are closely inspected for any evidence of neoplasm, or of persistent fistulae, and if necessary biopsies are taken. Finally the mobility of the lung itself is observed, for upon this treatment to some extent depends. In most cases the excursion with respiration is seen to be considerable and these require water sealed drainage at least for a time. In others mobility exists, but is much limited, and open drainage is satisfactory. In a few very chronic cases with greatly thickened pleura, the lung is quite immobile and decortication is then the treatment of choice. In most, then, when the cavity is quite clean, a wide-bored rubber tube is inserted into it, and the layers of the chest wall are stitched snugly about it. Side holes should not be cut in it, for granulations grow into them and bleed freely when the tube is removed. It is anchored to the skin by a nylon suture until the incision is healed, after which a safety pin is passed through its wall (so that the lumen is not obstructed) and is held flat and securely to the chest wall by two fairly wide strips of strapping (Fig. 24). The tube itself is passed through the lattice of the patient's back rest and connected with an underwater sealed drainage bottle containing a measured quantity of antiseptic fluids (Fig. 22). This fluid rises and falls with respiration as a meniscus in the long glass tube which delivers the drainage from the chest, and this excursion provides a measure both of the mobility of the lung and of the negative pull which is helping to re-expand it. Continuous suction is added to this influence by connecting the shorter outflow tube of the bottle to a suction pump in the ward.

These passive measures are supplemented by vigorous active breathing exercises, especial attention being paid to diaphragmatic movements and to the mobility of the chest wall and the patient is

instructed to devote at least ten minutes of every hour to practising them With each day that passes the lung should advance a little farther towards the chest wall, the pleural layers becoming adherent as it does so, and the volume of the empyema cavity correspondingly

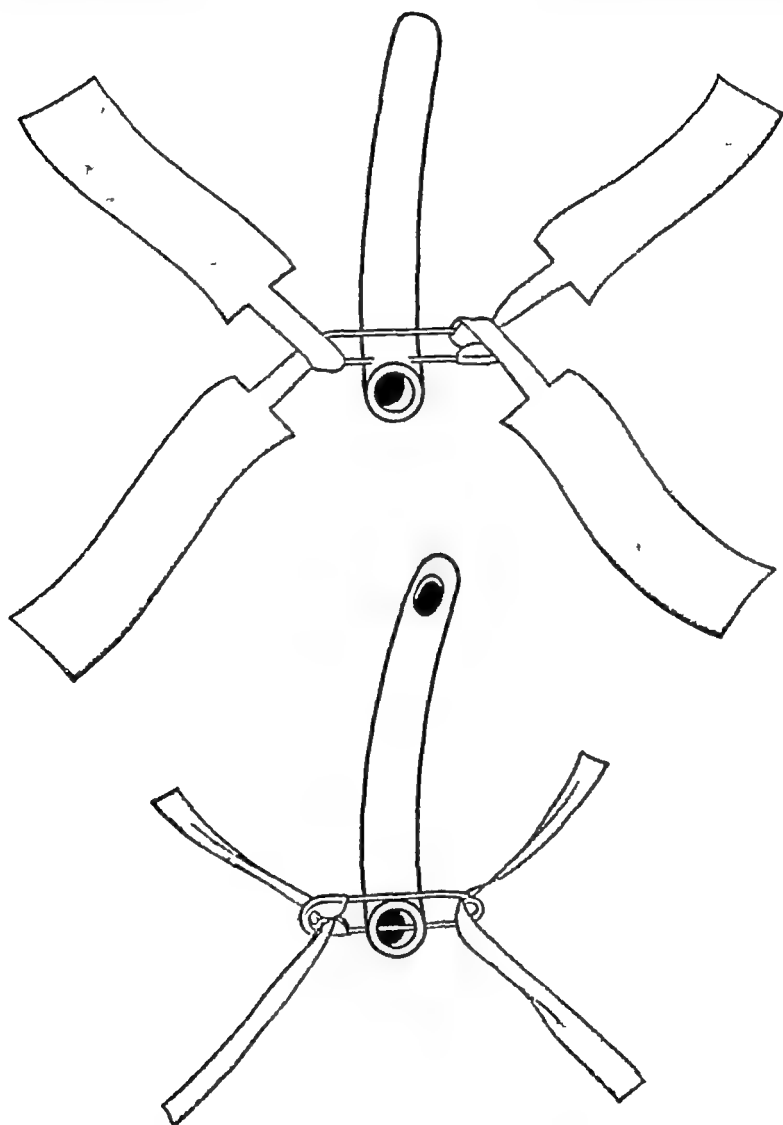


FIG. 24

Good and bad fixation of empyema drainage tubes In the bad the safety-pin occludes the lumen which will block with the first fragment of slough or fibrin; the tapes are inadequate, and do not hold the pin flat and securely against the chest, and a side hole has been cut into which granulations will grow and bleed when the tube is extracted Note how the strapping is arranged on the correct tube, and the position of the safety-pin (This drawing shows the tubes after open drainage has begun, and the water-seal disconnected)

diminishing. When a week or ten days has elapsed this progress will probably slow down, and the advantages of the underwater seal are then outweighed by the disadvantages of keeping the patient anchored to his bed by the apparatus. It is then disconnected, the tube cut short just beyond the retaining pin (Fig. 24) and allowed to drain directly into a dressing, and the patient got up and about, so that his exercises may be intensified. When he is fully active he can continue his treatment (which is now chiefly the management of his chronic empyema cavity) as an out patient.

4 Intercostal drainage

In small children or in elderly patients who are not well enough to move from their beds, an intercostal drain consisting of a self retaining catheter of the Malecot or de Pezzer type, is inserted into the chest by means of a large trocar and cannula passed through the most dependent intercostal space, after it has been infiltrated with local anaesthetic. The tube is afterwards connected to an underwater seal in the manner already described. This method, which has the advantage of being possible to carry out in the patient's own bed in the ward or at home, has two serious disadvantages firstly the lumen of the tube, which is limited by the dimensions of the cannula through which it must first be passed, is too small for adequate drainage in an adult, and too easily blocked by clots and secondly because no rib has been resected, the tube is tightly confined in the intercostal space and soon presses upon the intercostal nerve running along the inferior border of the uppermost rib so that it causes pain and is difficult for the patient to tolerate for very long.

Management

The commonest and most disastrous single mistake made in the treatment of chronic empyema is to remove the drainage tube too soon. It must not be taken out because the pus has become sterile or has ceased to drain or for any other reason whatsoever except that the lung has fully re-expanded and obliterated the whole empyema cavity. The tube track then extends merely through the thickness of the chest wall. When this stage has been reached, and not before it may safely be removed. If the tube is removed prematurely the chest wall rapidly contracts so that even after a few hours the same tube cannot be reinserted in the track. The latter may heal completely or leave a narrow sinus which periodically extrudes a bead of pus, but inside the chest a time bomb has been left which sooner or later perhaps in weeks, perhaps not for months or even years, will explode.

At the worst it kills the patient by flooding his bronchial tree with pus, or by giving rise to amyloid disease or to a cerebral abscess, at best it may cause repeated obscure febrile illnesses or track about in the pleural space, periodically presenting as a fluctuant swelling upon the chest wall breaking down in turn to form a fresh sinus

As soon as open drainage has been established the progress and shape of the empyema cavity is carefully watched by periodic pleurograms. At intervals of a few weeks the tube is removed, a soft rubber catheter is inserted into the track, and Lipiodol run in (X-ray 20). The track is lightly plugged with ribbon gauze, a radio-opaque marker is taped over the skin sinus, and X-rays are taken in a variety of positions so that the whole of the internal shape and dimensions of the cavity are clearly outlined. The plug is then removed, the Lipiodol run out, and the drain reinserted. Such a pleurogram may reveal that drainage is not truly dependent, and that several ounces of pus are in fact stagnating below the level of the drain (Fig. 25*b*); or that the lung has re-expanded in such a way that part of the cavity is being cut off from the drain, and a narrow isthmus formed through which it is necessary to pass a longer tube in order first to drain the distal pocket (Figs 25*c* and *d*). If the progress of the track is followed only by the passage of probes or bougies such deformities are missed, the end of the probe being arrested at the stricture and possibly a substantial cavity beyond going undetected. Such defects must be corrected: a rib lower down is resected to ensure truly dependent drainage; a stenosis dilated, or an unsatisfactory pocket drained afresh by the insertion of a separate tube. If dilatation of a track is necessary it must never be attempted with a solid dilator of the Hegar type or with a bougie which acts as a piston in a cylinder, compressing the air before it and creating a high positive pressure which may easily cause the death of the patient by air embolism. Such fatalities are quite common and are explained in coroners' courts as being due to 'pleural shock', a condition which is wholly mythical. In all cases a hollow sound with a perforated head is used, or if only the chest wall track is to be dilated, a laminaria tent is inserted and left *in situ* for 24 hours. Sharp or metallic probes ought never to be employed in examining chest sinuses, but a flexible gum-elastic bougie is used instead, whose diameter is less than that of the track.

Sudden cessation of discharge from the drain is a danger signal, especially if it is accompanied by a rise of temperature. It indicates that somewhere obstruction has occurred, either by bottle-necking

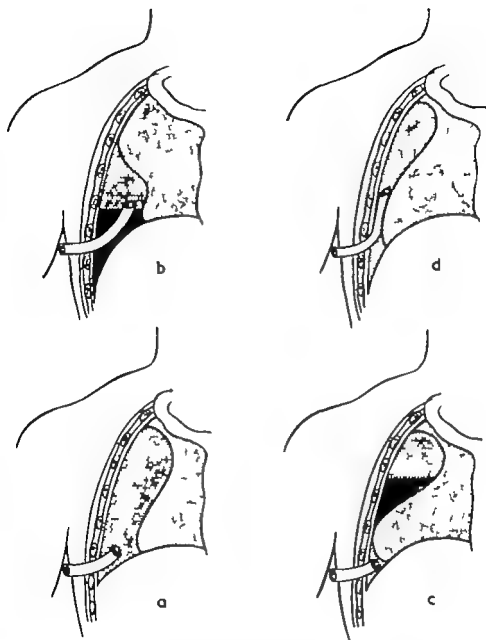


FIG. 25

- (a) A satisfactory chronic empyema drainage.
 (b) The tube is too long, allowing a puddle of pus to collect below it.
 (c) The lung has partially re-expanded, causing a bottle-neck of the empyema track. If the track is merely measured with a probe the length of the tube will appear satisfactory and the large loculus of pus above remain undrained.
 (d) The position in (c) corrected: the track has been dilated and the tube passed up it to drain the rest of the empyema.

of the track, or by blockage of the tube with slough or clot. If pus gushes out after the tube is removed the tube is either too long, and has been acting as a bung, or is itself obstructed. Aided by vigorous breathing exercises, the lung should advance steadily, progressively diminishing the cavity until it everywhere reaches the chest wall and virtually extrudes the drainage tube. Failure of the lung to do this must lead to a search being made for the cause. In addition to non-dependent drainage, or bottle-necking of the track, other possible impediments are: secondary infection of the area by tuberculosis, or actinomycosis; a growth, or a persistent fistula in the underlying lung, or the presence of a foreign body within the cavity.

Quiescent foci of *tuberculosis* may be rekindled by nearby disease in the lung, and retard normal healing. As a rule tubercle bacilli are not found in the pus from such an empyema sinus, but guinea pig inoculations or biopsy of the track revealing typical giant-cell systems, may lead to a diagnosis. A persistent bronchial fistula (X-ray 20) may also be due to this cause; and resection of the underlying lobe of the lung together with the empyema is necessary.

Actinomycosis is sometimes the primary cause of an empyema, when the characteristic fungi and their 'sulphur granules' can be demonstrated. It is treated by aspiration and penicillin, which is injected both into the cavity and intramuscularly in doses of 2,000,000 units a day continued for at least six weeks. *Actinomyces*, however, are not always pathogenic in the lung and sometimes appear as saprophytes and casual contaminants.

The commonest *foreign body* to find its way into the cavity is the drainage tube itself, for if the pin is removed at home, or if it accidentally becomes detached from the rubber, any sharp intake of breath or pressure against the tube results in its being sucked or pushed inside. If the patient is unaware of this, as he may be, the tube is replaced by another and the presence of the lost tube within the chest remains unsuspected, for unless a barium-lined tube has originally been employed, it will not be visible by radiography. Once the possibility has been envisaged, however, a thoracoscope or bronchoscope can be passed along the track, the interior of the cavity inspected, and the tube sometimes extracted without further surgery.

A *carcinoma*, or *adenoma*, in the underlying lung should be detected early in the disease by bronchoscopy, unless the growth lies beyond bronchoscopic vision. Adenomas are seldom, if ever, so peripheral; but about 30 per cent. of carcinomas are. In these cases inspection and biopsy at the time of drainage should reveal the

tumour but if these investigations have been negative a further biopsy is taken from the interior of the track.

In some rare instances *infected congenital cysts* are mistaken for empyemas and drained. As the cyst wall remains the cavity will not close, and until the true diagnosis is made, cure—by excision of the whole cyst—is impossible. The case history of the child with a sequestered pulmonary segment given on page 129 is instructive and relevant (X rays 31 and 35)

THE CHRONIC EMPYEMA

A long-standing chronic empyema is a monument either of neglect or of surgical incompetence. The acute empyema has either not been diagnosed at all, or has been treated ineffectively. The longer it has been present the greater the ensuing deformity of the chest and the greater the corresponding impairment of function. The body attempts, in its response to infection to perform a physiological thoracoplasty: the ribs fall together becoming more and more oblique and triangular in section; the diaphragm rises in the thorax and is immobile, the heart and mediastinum are dragged to the side of the lesion instead of being pushed away from it; the lung is incarcerated and functionless, scoliosis develops reducing still more the size of the hemithorax. The accessory muscles of respiration waste, and as these also move the shoulder that joint is limited in range stiff and painful. Clubbing of the fingers and pulmonary osteoarthropathy may be present, and sometimes amyloid disease with albuminuria, oedema, and hepatic enlargement. If a rib resection has previously been performed, it is likely that, either at the site of operation, or elsewhere upon the chest wall, a narrow sinus will be found from which oozes an occasional bead of pus.

Under such circumstances the first thing to do is to bronchoscope the patient and exclude the presence of a carcinoma, an adenoma, or other bronchial obstruction. The sputum is examined for tubercle bacilli, for malignant cells, and to determine the nature of the predominant organisms. If a bronchial fistula exists, the patient coughs up the contents of the empyema, and a fluid level is therefore visible within it on radiography. If he is coughing up pus but no level is visible, he probably has related bronchiectasis or suppurative pneumonia which can be demonstrated by bronchography. When disease of this kind is present in the lung there is nothing to be gained by attempts to redrain the empyema: the correct treatment is lobectomy or pneumonectomy according to the position and extent of the lesion, combined with pleurectomy and the removal of the whole

infected mass If only lobectomy is required, the remaining lobe is carefully decorticated and every effort made to re-expand it and thereby fill the chest. After pneumonectomy for non-malignant lesions a thoracoplasty is performed, obliterating the space and preventing over-distension of the opposite lung

When an underlying lung, however, is apparently healthy, adequate drainage of the empyema must be established If a sinus exists it is dilated with a laminaria tent until an adequate drainage tube can be inserted, and pleurograms carried out If the existing sinus proves not to be dependent, or if none at all exists, Lipiodol is injected to mark the bottom of the cavity and a new and more suitable rib resection performed The interior is then inspected for growth, for fistulae, for rib sequestra, and for foreign bodies such as portions of rubber tubing, and the remaining mobility of the lung carefully observed. The pus itself is examined for tubercle bacilli, for actinomyces, and for other organisms *If the lung retains some mobility* adequate drainage in good position combined with correction of posture and intensive breathing exercises will effect a cure, though the length of treatment needed is proportionate to the duration of the disease, the longer pus having been present in the pleural cavity the thicker and more rigid being the carapace of fibrin deposited upon the lung and the chest wall

If there is little or no mobility of the lung it is better not to waste time with prolonged and wearisome treatment and uncomfortable drainage tubes, but to proceed at once, providing the patient's general condition permits, with thoracotomy and decortication of the imprisoned lung The technique of the latter operation is fully described in the section on tuberculous empyema

It is doubtful whether much room is left in modern therapy for the mutilating operations of Roberts or of Schédé. They are based, as is the use of thoracoplasty in such cases, on the proposition that if the lung cannot be expanded to the chest wall the chest wall must be brought down to the lung by the removal of ribs However, if no lung exists to fill the space (as for example when an empyema follows bronchial fistula after pulmonary resection), a *thoracoplasty* is of value both in diminishing the dead space and in bringing the thoracic wall into contact with the bronchial stump and so helping to seal it off *Schédé's operation* consists essentially in unroofing a very small rigid empyema cavity, saucerizing it, and allowing it to heal by granulation. The overlying ribs are resected and all the external tissues cut away with a diathermy loop so that there is no overhang at any point. The cavity is then lightly packed with *tulle gras*

When the granulating surface is clean healing is expedited by skin grafting. For rather larger residual empyemas with rigid walls *Roberts operation* employs a similar principle. The drainage track is excised, the cavity opened, and after the skin and muscles covering it have been retracted, the overlying ribs are resected subperiosteally. The parietal layer of thickened pleura and scar tissue is then cut away so that the skin, muscles and periosteum form a soft flap hinged on the side of its blood supply, to fall in against the visceral surface of the cavity and adhere to it. A pack, placed externally by some, subcutaneously by others retains it in this position for 8-10 days and is then removed, by which time healing should have taken place. Both *Schédé's* and *Roberts operations* can lead to considerable blood loss and a serious disadvantage shared by both is the probability that several intercostal nerves may be damaged with resulting paresis and anaesthesia of a part of the abdominal wall.

ABSCESSSES OF THE LUNG

THE MALIGNANT ABSCESS

IN older textbooks, and consequently in the forefront of many doctor's minds, the only condition to be found under the title 'lung abscess' is the acute, putrid, aspiration kind; but in fact this is only one of many varieties of lung abscess, and of them all the malignant type (i.e. an abscess associated with a carcinoma) is not only the most important but by far the commonest. So much is this the case that *any cavitated lesion in the chest X-ray of a man over 40 demands a provisional diagnosis of bronchial carcinoma*, with full investigation, including bronchoscopy, until the presence of a growth is disproved. Naturally such a cavity may be tuberculous, in which case other evidence of tuberculosis in the lung is likely, and tubercle bacilli will almost certainly be present in the sputum; but weeks and months spent searching fruitlessly for them may cost the patient his life if in fact the underlying lesion is a neoplasm.

A greengrocer aged 47, the father of three small children, was referred to a Chest Clinic after having had an haemoptysis. X-ray of his chest showed a small cavity containing a fluid level near the apex of his right lung. His sputum was tested for tubercle bacilli but none were found; and he was told to come back in one month for further tests. The sputum was again negative, the cavity a little larger, and he was sent to a small sanatorium where numerous sputum tests were repeated with the same result. In spite of this, however, he was given streptomycin and an attempt was made to induce an artificial pneumothorax. Subsequently his right phrenic nerve was crushed in the neck. The cavity continued to increase slowly in size until, almost a year after its initial discovery, he was referred with the suggestion that the cavity seemed suitable for thoracoplasty as it had not responded to prolonged bed rest. It was a malignant abscess, and squamous carcinoma cells were present in his sputum. At thoracotomy many secondary deposits were found in the mediastinal glands, and the patient died of them soon after. Twelve, or even six, months earlier the growth would have been readily operable, and curable.

There are three kinds of malignant abscess, the first very common, the other two less frequently encountered but still by no means rare:



17

-ray 17. A generalized empyema in the right pleural cavity displacing the heart to the left. It was purulent and antibiotic. (See X-ray 18.)



18

-ray 18. After aspiration alone of the empyema seen in X-ray 17 the right lung has fully re-expanded. Extirpation was necessary.



19

X-ray 19. A posterior basal empyema limited by pleural adhesions. It is of great importance not to extend to the rest of the pleural cavity.



20

X-ray 20. A pleurogram performed after dependent drainage of an empyema. The lung has re-expanded completely. The residual empyema cavity is small but Lipiodol has entered the bronchial tree proving bronchopleural fistula still exists.

1 The first variety is that in which the centre of the growth, almost always a squamous-celled carcinoma, becomes necrotic and breaks down to form an abscess (X rays 49 50 55) Such a cavity from the nature of its origin is likely to be eccentric and irregular, and to have thick and ragged walls consisting of the growth itself The X ray appearances are quite characteristic, and a positive diagnosis including the histology of the neoplasm can usually be made on sight. Malignant cells are often present in the sputum to provide confirmatory evidence It is, however, in those cases where the abscess is small, and its walls therefore relatively more regular that mistakes are most likely to be made and the abscess mistaken for, and treated as, one of the aspiration type The absence of any history to suggest such an origin, the persistence of the lesion after a brief course of antibiotic therapy and the irregularity which will be revealed in more detail by tomography, should all put the clinician on his guard but this guard will never have been relaxed if he remembers that such an abscess is in any case most likely to be due to cancer if the patient is male, and over 40 I have repeatedly seen such lesions treated with antibiotic after antibiotic for long periods before their true nature was suspected. Fortunately as most are well-differentiated squamous growths, they often remain operable even at the end of much lost time

A man aged 65 was admitted to a general hospital with a small abscess cavity in the upper lobe of his right lung. The organisms in his sputum were cultured and he was treated with all the antibiotics to which they were in turn proved sensitive, first penicillin then streptomycin, followed by aureomycin Chloromycetin, Terramycin and erythromycin During prolonged treatment with the latter drugs he suffered severely from diarrhoea due to ulceration of his bowel nearly costing him his life and greatly worsening his condition In all, he spent fifteen months in hospital and had innumerable X ray films taken, all of which were meticulously reported upon, minor changes in the persistent abscess cavity being described. At the end of fifteen months the growth proved still to be operable by lobectomy The patient returned home in two weeks and remains well. Excluding the cost of his operation to the state at present rates only a few shillings, I calculated that the failure to make a correct diagnosis had cost the taxpayer well over £1 000 and the man himself was fortunate to escape with the loss merely of fifteen months of his life rather than the whole

2. The second type of malignant abscess is simply the consequence of bronchial obstruction by the neoplasm, which may itself be quite

small. If acute infection occurs distal to such a blockage and causes an abscess, the latter may resemble in all respects, save in the history of a cause, an ordinary aspiration abscess, or one occurring distal to an inhaled foreign body. The cavity is symmetrical, its wall is regular (X-rays 51, 52), the pus coughed from it may be offensive, and it is quite likely to resolve when antibiotics are given and the inflammatory reaction round the growth itself subsides sufficiently to permit regular drainage into the bronchus. As a result the X-rays may show such resolution that the malignant origin of the abscess will be missed if bronchoscopy is not performed, as of course it must be in all such cases.

A publican was admitted to a thoracic surgical unit with fever and foul expectoration from what looked like a simple aspiration abscess in the apex of his right lower lobe. As this diagnosis seemed to be confirmed by a history of having 'passed out' during a recent drinking bout he was treated with penicillin. In ten days the abscess had completely melted away and all expectoration ceased. He was discharged *without having being bronchoscoped*. Three months later the small carcinoma which had been present all the time at his right hilum was inoperable.

3 The third and last variety of malignant abscess is the least common and the most treacherous. It is the 'spill-over' abscess caused by inhalation into some other, dependent, part of the lung of a fragment of pus or necrotic growth from a neoplasm elsewhere which may be so small that it passes unnoticed. It will be seen that this is in fact an 'aspiration' abscess, indistinguishable from any other pyogenic aspiration abscess except in the absence of a suggestive history, and unless the possibility of a related growth being present is kept in mind, the neoplasm is likely to be missed.

Some time ago I saw a patient with very foul teeth and gums who had a typical aspiration abscess in one of the axillary segments of his right upper lobe. A week of penicillin resulted in complete clearing of his chest X-ray, and after having his teeth attended to, I very reprehensibly allowed him to leave hospital without his being bronchoscoped. Fortunately he attended the follow-up clinic a month later, and I was then horrified to find he had complete collapse of his right lower lobe. Immediate bronchoscopy confirmed that this was due to a growth, and a right pneumonectomy followed; but my carelessness might easily have cost him his life.

Another patient with fever, pleurisy and profuse stinking sputum was admitted, with an aspiration abscess in the apex of his left lower lobe (X-ray 53). Swift resolution followed antibiotic therapy,

bronchoscopy was performed and was normal, his X ray seemed clear, and he was discharged. A few weeks later in the follow up clinic he reported himself completely cured, but a very small abnormal opacity was noticed in the upper lobe of his right lung (X ray 54). A review of the earlier films confirmed that it had been present all the time the abscess was certainly a 'spill-over' one, and the opacity in the opposite lung a carcinoma. Although sputum tests and bronchoscopy were quite negative a right upper lobectomy was performed at once, and the diagnosis confirmed.

THE ASPIRATION ABSCESS

We all aspirate foreign fragments into our lungs, but are usually able to cough them out again and the ciliary action of the columnar cells lining our bronchi help to eliminate microscopic particles. If the cough reflex is interfered with, however, by anaesthesia, by drugs by drink, or by any form of deep unconsciousness or coma, matter once inhaled is not coughed up but becomes lodged in a bronchus, whose size depends upon the size of the fragment and whose position in the lung depends upon the posture of the patient at the time of inhalation. The fragment thus causes obstruction and therefore collapse of the aerated lung distal to it. If this bronchial embolus is highly septic (e.g. a flake of dental tartar or a fragment of tonsillar debris) it sets up an intense inflammation of the surrounding tissue which suppurates, sloughs and finally forms an acute abscess cavity. The exact course of the illness and the subsequent character and behaviour of the abscess are modified by the resistance of the patient and the nature of the infecting organisms. Resistance is lowered by exposure and immersion and many lung abscesses were seen during the Second World War in air-crew and seamen who found themselves for long periods in the water and had aspirated some of it, even though they had not actually lost consciousness and could still cough. In others foreign material may enter the lung and remain there for a considerable time without causing an abscess simply because no virulent organisms happen to accompany it, or the resistance of the patient is high.

The acute putrid lung abscess is putrid (i.e. productive of foul stinking pus) because it is heavily infected by anaerobic streptococci from the mouth, and is the classical form of the aspiration abscess but it must be remembered that any suppurating lung cavity whatever its origin, becomes foetid if infected with anaerobes, whereas occasionally an aspiration abscess is free of them, and the pus from it is therefore not offensive.

Aspiration abscesses depend upon two things:

- (1) the impairment of the cough reflex;
- (2) the consequent inhalation of septic material, the latter resulting in bronchial obstruction and infection.

It will be noted that these conditions are perfectly fulfilled during operations under anaesthesia on the upper respiratory tract, such as tonsillectomy, adenoidectomy and tooth extractions, and that in all these there is an abundance of highly septic material, gland and mucosal fragments, pus and infected blood clot, at hand. In this country tonsillectomy is almost always performed under good general anaesthesia, with the patient's head at a lower level than his lung, so that aspiration abscesses following it are consequently exceedingly rare; but elsewhere it is done under local anaesthesia with the patient sitting upright in a chair, and postoperative lung abscesses therefore occur. In marked contrast, dental extractions are habitually performed in England with the patient sitting upright, and as these are often multiple and sometimes carried out under hasty and not very skilful general anaesthesia, it is only surprising that aspiration abscesses are not more common. Clearly similar risks attend anaesthesia for any operation, and are the chief reason why an early return to consciousness and the ability to cough is so desirable. Postoperatively the trachea should be aspirated, and if necessary a bronchoscope passed. With modern anaesthesia and the avoidance of overdosage with drugs like Pentothal, consciousness is often regained in the theatre even after long operations, and the patient is encouraged at once to cough. If he is still unconscious on return to the ward he should never be allowed to lie flat on his back, but be placed semi-prone, with the head turned to one side, and the foot of the bed slightly raised so that aspiration of blood or mucus cannot occur. A nurse sees that a good airway is maintained, and the nose and mouth kept clean. Coughing is begun as soon as possible and helped by supporting the patient's wound manually and giving him enough drugs to keep him relatively free from pain, but not enough to depress his cough reflex. Any subsequent evidence of pulmonary collapse is an indication for prompt bronchoscopy with aspiration.

In dental surgery multiple extractions should never be performed upon a patient sitting in a chair, and anaesthetics should always be given under good conditions by skilled practitioners; nor does local anaesthesia confer immunity, for anything which interferes with pharyngeal sensation favours inhalation. Real safety is to be secured

only by keeping the patient's head at a lower level than his thorax, on an operating table

Even after operations unconnected with the upper respiratory tract, the source of lung infection is often decayed teeth covered with septic scales of tartar and accompanied by gums running with pus, and the passage of intratracheal tubes through such a mouth is dangerous. In a group of 318 abscesses not due to malignancy Brock found 68 (21.4 per cent.) to be postoperative, 22 of them following teeth extractions and Stern in 115 cases attributes 16 per cent. to tonsillectomies, 11.3 per cent. to dentistry and notes that 84 per cent. of the whole group had bad teeth, tartar encrusted, and infected gums. It is, in short, unusual to encounter an aspiration abscess in a patient who has not got bad teeth and the same relationship exists between dental sepsis and chronic suppurative pneumonitis. It is better to prevent abscesses than to cure them and attention to diseased teeth and gums is an indispensable preliminary to any major operation.

When anaesthesia has not in fact preceded the development of an aspiration abscess, some other reason for suppression of the cough reflex must be sought, such as a history of epilepsy or diabetic coma, of head injury or drug taking, of partial drowning, or of alcoholism. An explanation of this kind can be elicited in about two-thirds of the patients but when it cannot, secret alcoholism irrespective of the sex or condition of the patient, should be considered as well as the serious possibility that the abscess may not after all be of the aspiration type or that it is associated with a focus of pulmonary sepsis elsewhere which is less conspicuous radiographically but from which it has spilt over.

The situation of an aspiration abscess in the lung is determined firstly by the bronchial anatomy and secondly by the position of the patient at the moment of inhalation. The right stem bronchus more nearly continues in the line of the trachea than does the left, and so is the more likely first to receive an inhaled septic embolus. If the patient is lying on his back, the first bronchial orifice such a fragment, or bead of pus, meets as it rolls down the main bronchus, is that to the apical segment of the lower lobe but if the patient is lying on the right side, the upper lobe branch is dependent, and the embolus is then likely to find its way into the posterior segment, or into one of the axillary branches, of the upper lobe bronchus. Should the patient happen to be inclined to the left, the left main bronchus is entered, and thence the apical segmental bronchus of the left lower lobe or if wholly on his side the left upper lobe bronchus. The distribution of aspiration abscesses is thus explained, and their embolic

origin confirmed. It is interesting to note that these are also common sites for tuberculous cavitation. So unusual are deviations from this pattern that one must hesitate before diagnosing an aspiration abscess in any other pulmonary segment. I remember a patient who had a small abscess in the anterior segment of his right upper lobe. It was so peripheral that I was inclined to regard it as a carcinoma, but examination showed him to have particularly foul teeth, and a careful history elicited the fact that a week previously he had made a long journey in very wintry weather on the pillion seat of a motorcycle, crouched forward and with a heavy pack on his back. His posture thus amply explained the situation of the abscess in the anterior segment, and his long exposure and septic teeth provided a satisfactory aetiology.

The size of the septic embolus determines the size of the bronchus in which it lodges, but usually this is a segmental or subsegmental one, and the consequent atelectasis extends wedge-like to the pleural surface. The airless parenchyma is rapidly invaded by the accompanying organisms, which, as they have mostly originated in the mouth, are likely to include fusiform bacilli, spirochaetes and anaerobic streptococci. Coliform bacilli are also commonly present, and by no means imply that the lung infection is secondary to bowel involvement or to extension of a subphrenic abscess. The resulting area of intense pneumonitis in the segment soon undergoes suppuration, and necrosis leads to the formation of an abscess cavity full of stinking pus. As it increases in size, occupying more and more of the affected segment, it approaches the pleural aspect of the lung, but the subpleural vascular plexus helps to stem the gangrenous advance, and for a time at least, a layer of pulmonary tissue is preserved between the abscess and the visceral pleura.

Because these developments take some time, there is usually a 'latent period' of a week or ten days between the operation, or other event which led to the aspiration, and the onset of symptoms, although this period varies with the virulence of the infection and the size of the obstruction. It is generally marked by a rigor, with rise of temperature, followed by pleural pain. These symptoms are often dismissed simply as 'pleurisy' or 'postoperative pneumonia'; and if antibiotics are given they may be masked, and the abscess is either aborted, or passes undiagnosed into a chronic state.

At first the patient has little or no sputum for no drainage is taking place in a bronchus. He is ill, toxæmic, and runs a hectic temperature. The sputum is brown and coated. There are dullness, and crackles over the affected part of his chest, dullness,

lessened air entry and sometimes pleural friction. The white cell count rises to 20,000 or more and postero-anterior and lateral X rays reveal a rounded homogeneous opacity with ill-defined margins, usually situated in the posterior or axillary part of an upper lobe or in the apical segment of a lower

After a day or two a small haemoptysis occurs followed by sudden fetor, a foul taste, and, as the abscess finally ruptures into the adjacent bronchus, expectoration of copious stinking pus, the offensive nature of which sometimes makes it difficult to approach the patient. Several ounces of pus are coughed up daily and in this way natural drainage of the abscess occurs, often resulting in progressive diminution in its size, and spontaneous cure, although there may be residual scarring or bronchiectasis of the affected segment. As soon as rupture into the bronchus takes place a fluid level appears in the X ray opacity, identifying it beyond any doubt as an abscess even if the more severe symptoms have been masked by antibiotics (X rays 22 and 23).

Although it is the rule, resolution of this kind depends upon early bronchial drainage and this cannot be relied upon. If it is delayed the abscess continues to enlarge until it invades the protective outer lung cortex, which becomes necrotic as its vessels thrombose and the abscess reaches the visceral pleura, rupturing through it and producing an empyema. This disaster is preceded by the development of a pleural effusion and the appearance of such fluid in the pleural space provides a warning that an empyema is not far off.

Even though pus is apparently being coughed up freely drainage may in reality be inadequate and should it suddenly cease, and the temperature rise, the situation at once becomes dangerous. Sometimes a slough within the abscess cavity acts as a ball-valve admitting air on inspiration but falling back over the orifice on expiry preventing drainage, so that the cavity rapidly enlarges and the same mechanism comes into play if the fistula between abscess and bronchus is narrow and oblique. With every increase in size of the cavity this obliquity too is increased and its effect accentuated. If rupture into the pleural space does not soon follow the abscess extends by direct invasion of the surrounding lung, new diverticula opening out of the parent cavity until the whole parenchyma is honeycombed by them. It is this state of affairs that one finds established in chronic suppurative pneumonia.

Another mode of extension is by septic bronchial embolism, the same mechanism whereby the abscess itself began. Some pus or a fragment of slough, spills over and is aspirated into a dependent

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bronchus, setting up acute pneumonitis in the collapsed lung segment, and creating a new abscess. This tendency for acute aspiration abscesses to 'migrate' is also a feature of chronic suppurative pneumonitis, and it is not uncommon when an abscess presents first in the right upper lobe to see propagated, a few days after drainage from it begins, a further abscess in the apical segment of the lower lobe or even in the opposite lung.

So long as any collection of pus in the lung remains inadequately drained there is also grave risk of a metastatic cerebral abscess, and this in itself should be enough to impart a sense of urgency to treatment.

Finally, the longer an abscess remains, and the less efficient its drainage, the more likely is it to enter a chronic phase which can be as dangerous as the acute, or at best, a source of lingering ill-health. This is especially apt to occur today when the initial symptoms of an abscess are often largely suppressed by antibiotics and it remains undiagnosed. In some, the abscess diminishes in size but does not wholly resolve, leaving a small chronic cavity into which epithelium grows from the communicating bronchus, lining it with squamous or cuboidal cells. Once this has happened it can never close, but persists, like a chronically infected cyst, a source of purulent sputum.

In others, chronic suppurative pneumonitis with extensive honeycombing of the lobe results, and the volume of pus expectorated may be as much as half a pint a day. After any acute abscess, even if resolution has been prompt and complete, it is common to find that permanent damage has been done to the bronchi of the affected segment so that a patch of localized bronchiectasis remains.

Treatment

Antibiotics have transformed the treatment of aspiration lung abscesses. Prior to their use nearly all required open surgical drainage, and both the mortality and subsequent morbidity were very considerable. Although drainage is now rarely indicated, the conservative management of an abscess is a surgical problem in the chest as it is in the abdomen, and not a medical one. The attention of the surgeon must be constantly directed to securing prompt and early resolution and to preventing an acute abscess becoming a chronic one, for once chronic suppuration is established in the lung permanent damage results which can never be repaired, and is likely to prove fatal unless it is removed.

As soon as a diagnosis is made, and the abscess accurately local-

ized on postero-anterior and lateral X rays penicillin therapy is begun in doses of 500 000 units six hourly. If examination of the sputum reveals penicillin-resistant organisms their sensitivity to other drugs is determined and the appropriate antibiotic substituted. Bronchoscopy should be carried out so that the involved segmental bronchus can be inspected and any slough or debris interfering with drainage from it removed at the same time the presence of a neoplasm or an inhaled foreign body is excluded. There is no value in aspirating pus from an abscess at bronchoscopy, and the insufflation of antibiotics is a dangerous and useless practice.

Postural drainage is prescribed as soon as the anatomical position of the abscess in the lung is accurately known, and should be continued for periods as long as the patient will tolerate. The physiotherapist helps by encouraging coughing during these, and by slapping and percussion over the related chest wall. Sometimes it is found that a change of position or even a posture different from that indicated by the bronchial anatomy proves more productive of sputum and should therefore be employed. Whatever measures drain most pus, are best.

The abscess should now rapidly and progressively diminish in size and routine chest X rays are taken every two or three days in order to see that this is happening and that no impediment to drainage has developed, as would be suggested by increase in the size of the cavity or by a persistent fluid level, or by sudden diminution or even cessation of the sputum, with fever. Any of these signs call in the first instance for re bronchoscopy when it is often found that a piece of slough is blocking the bronchus and can be removed with grasping or biopsy forceps but should this not be the case and drainage remains unsatisfactory direct surgical intervention becomes necessary.

This takes one of two forms: rib resection and the direct insertion of a drainage tube into the abscess cavity, or primary resection of the affected segment. The first of these once the usual treatment for all such abscesses is now very rarely employed but it may be needed in acute cases where a slough within the cavity is preventing bronchial drainage, or in patients so ill, or so old and frail that primary resection is out of the question. It should never be used when the abscess has become chronic (i.e. after the first few weeks of illness) for by that time the cavity walls have become rigid and infiltrated, satellite abscesses may have formed, bronchial fistulae become established, or even secondary infection occurred from reactivated tuberculous lesions nearby making primary closure and healing of

bronchus, setting up acute pneumonitis in the collapsed lung segment, and creating a new abscess. This tendency for acute aspiration abscesses to 'migrate' is also a feature of chronic suppurative pneumonitis; and it is not uncommon when an abscess presents first in the right upper lobe to see propagated, a few days after drainage from it begins, a further abscess in the apical segment of the lower lobe or even in the opposite lung.

So long as any collection of pus in the lung remains inadequately drained there is also grave risk of a metastatic cerebral abscess, and this in itself should be enough to impart a sense of urgency to treatment.

Finally, the longer an abscess remains, and the less efficient its drainage, the more likely is it to enter a chronic phase which can be as dangerous as the acute, or at best, a source of lingering ill-health. This is especially apt to occur today when the initial symptoms of an abscess are often largely suppressed by antibiotics and it remains undiagnosed. In some, the abscess diminishes in size but does not wholly resolve, leaving a small chronic cavity into which epithelium grows from the communicating bronchus, lining it with squamous or cuboidal cells. Once this has happened it can never close, but persists, like a chronically infected cyst, a source of purulent sputum.

In others, chronic suppurative pneumonitis with extensive honeycombing of the lobe results, and the volume of pus expectorated may be as much as half a pint a day. After any acute abscess, even if resolution has been prompt and complete, it is common to find that permanent damage has been done to the bronchi of the affected segment so that a patch of localized bronchiectasis remains.

Treatment

Antibiotics have transformed the treatment of aspiration lung abscesses. Prior to their use nearly all required open surgical drainage, and both the mortality and subsequent morbidity were very considerable. Although drainage is now rarely indicated, the conservative management of an abscess is a surgical problem in the chest as it is in the abdomen, and not a medical one. The attention of the surgeon must be constantly directed to securing prompt and early resolution and to preventing an acute abscess becoming a chronic one, for once chronic suppuration is established in the lung permanent damage results which can never be repaired, and is likely to prove fatal unless it is removed.

As soon as a diagnosis is made, and the abscess accurately local-

ized on postero-anterior and lateral X rays, penicillin therapy is begun in doses of 500 000 units six hourly. If examination of the sputum reveals penicillin resistant organisms their sensitivity to other drugs is determined and the appropriate antibiotic substituted. Bronchoscopy should be carried out so that the involved segmental bronchus can be inspected and any slough or debris interfering with drainage from it removed, at the same time the presence of a neoplasm or an inhaled foreign body is excluded. There is no value in aspirating pus from an abscess at bronchoscopy, and the insufflation of antibiotics is a dangerous and useless practice.

Postural drainage is prescribed as soon as the anatomical position of the abscess in the lung is accurately known and should be continued for periods as long as the patient will tolerate. The physiotherapist helps by encouraging coughing during these, and by slapping and percussion over the related chest wall. Sometimes it is found that a change of position or even a posture different from that indicated by the bronchial anatomy proves more productive of sputum and should therefore be employed. Whatever measures drain most pus, are best.

The abscess should now rapidly and progressively diminish in size and routine chest X rays are taken every two or three days in order to see that this is happening and that no impediment to drainage has developed, as would be suggested by increase in the size of the cavity, or by a persistent fluid level, or by sudden diminution or even cessation of the sputum, with fever. Any of these signs call in the first instance for re-bronchoscopy, when it is often found that a piece of slough is blocking the bronchus and can be removed with grasping or biopsy forceps but should this not be the case and drainage remains unsatisfactory, direct surgical intervention becomes necessary.

This takes one of two forms: resection and the direct insertion of a drainage tube into the abscess cavity, or primary resection of the affected segment. The first of these, once the usual treatment for all such abscesses, is now very rarely employed, but it may be needed in acute cases where a slough within the cavity is preventing bronchial drainage or in patients so ill, or so old and frail that primary resection is out of the question. It should never be used when the abscess has become chronic (i.e. after the first few weeks of illness), for by that time the cavity walls have become rigid and infiltrated, satellite abscesses may have formed, bronchial fistulae become established or even secondary infection occurred from reactivated tuberculous lesions nearby, making primary closure and healing of

the cavity less likely As time goes on, epithelialization of part or all of it renders it permanent; and, at the very least, residual bronchiectasis of the segment is probable, and will demand further surgery For these reasons, therefore, such cases should be treated rather by primary resection of the involved lobe or segment, as this cuts short what may otherwise be a long and dangerous illness.

External drainage

Almost all abscesses soon become closely related to the visceral pleura, and are readily accessible somewhere on its costal aspect. It is therefore of prime importance to localize the abscess accurately by postero-anterior and lateral X-rays so that the appropriate rib for resection can be selected by counting the ribs from above down, and by measuring the distance out from the vertebral column. If conditions permit, and the abscess is situated posteriorly, it is safest to operate with the patient sitting upright upon a stool, and resting the head and arms forward upon the operating table, so that pus cannot flood into the opposite lung as might happen were he to lie on his side with the abscess uppermost About 3 inches of the rib most closely related to the abscess is resected in exactly the same way as when an empyema is drained (see p 87); the intercostal bundle is divided between ligatures, and the posterior layer of periosteum incised If localization has been accurate the parietal pleura thus exposed will be adherent to the visceral layer, and thickened and opaque Should it not be so the approach is probably inaccurate and must be revised, either by removing more of the same rib, or a portion of the rib above or below. Occasionally however the pleura remains free, and if this is the case the layers are stitched carefully together An aspirating needle is passed through the pleurae into the abscess, confirming its position, and the intervening tissue cut away with a diathermy loop The interior is inspected, and all slough and debris removed from the main cavity and from any extensions that are revealed A lanolin gauze-roll pack is then loosely inserted, and can be replaced after three or four days by a large-bore rubber tube draining directly into the dressings This should not impinge on the abscess wall lest it cause ulceration, with fistula formation or secondary haemorrhage; and it must not have a side hole cut into which granulations grow, so that bleeding occurs when it is removed. With continued chemotherapy and breathing exercises the cavity should shrink rapidly, its progress being checked by occasionally running Lipiodol into it and taking X-rays. From time to time the tube

requires shortening, until at last it extends only through the chest wall, and can be removed.

The chief complications of the operation are

(1) Secondary bleeding, generally from an intercostal artery but sometimes from erosion of a pulmonary vessel or from oozing granulations. Should this occur the patient must be taken to the theatre, where the interior of the cavity can be inspected in a good light and the trouble dealt with by ligation or coagulation.

(2) Empyema due to contamination of the pleural space. Any effusion must be completely aspirated and an empyema dealt with as described elsewhere.

(3) Bronchial fistula due to ulceration. As a rule fistulae soon close unless secondary infection with tuberculosis has occurred, or unless there is an underlying neoplasm. Biopsy of the track, bronchoscopy and a search for acid fast bacilli and neoplastic cells must be undertaken.

The presence of a persistent fistula, or the failure of an abscess cavity to heal completely is an indication for resection of the area.

CHRONIC ASPIRATION ABSCESES

The manner in which a residual abscess cavity can become epithelialized from the neighbouring bronchus and therefore perpetuated has already been described and only excision of such a chronically infected space will effect a cure for the patient spits up a little yellow pus from it so long as it remains, his symptoms closely resembling those seen in bronchiectasis which indeed may also be present.

A much more serious condition is that of *chronic suppurative pneumonitis* often described as though it were a new and exotic disease, but which is in reality simply a more extensive and diffuse form of aspiration abscess in a chronic state. We have already seen that if such an abscess fails to drain properly or if its acute phase is masked, and the more virulent organisms in it suppressed by antibiotics, it may smoulder on extending gradually into the neighbouring lung tissue by burrowing out new cavities, rather than exploding catastrophically as does the untreated and undrained acute putrid abscess. The longer it remains the more extensive the area affected until perhaps a whole lobe is honeycombed and destroyed. With time it also becomes fibrotic and epithelialized so that chemotherapy can never cure it, although it may temporarily reduce the chronic infection and so diminish the volume of sputum. The latter may be very great, for the chain of cavities come to occupy a considerable volume, and it is not uncommon to find such patients coughing up

as much as half a pint of pus a day. In addition, the eroded and ulcerated lung may bleed, giving rise to periodic haemoptysis.

As happens with acute abscesses, so with chronic suppuration, debris or slough are often aspirated into other parts of the lung causing new 'spill-over' areas of infection resembling the parent lesion; for the organisms, and the patient's resistance to them, remain the same, and a non-offensive chronic abscess in one place seldom leads to an acute putrid abscess in another. The course is indolent because the virulence of the infection is low, and it is common to see one patch apparently burn itself out as a new area flares up, only to die down in its turn with perhaps a recrudescence in the original site. Wherever the infection becomes quiescent it leaves behind fibrosis and destruction of the normal lung tissue; and if it remains untreated the disease is likely to end fatally, even though the ever-present danger of a metastatic cerebral abscess is avoided, or the patient does not fall victim to amyloid disease.

Sometimes a typical history of an acute aspiration abscess can be obtained, or at any rate the patient remembers some period when the taste or smell of his sputum was foul, but if anaerobes have never been present, or have been suppressed, no such history is elicited, and the onset is perhaps diagnosed as bronchopneumonia. It is significant, however, that this episode will often be found to have followed dental extractions, or an operation, or indeed any of those events commonly associated with acute aspiration abscesses; and gross dental sepsis is nearly always to be found on examination.

Apart from purulent expectoration the patient complains of recurrent attacks of 'pneumonia' and pleurisy, a reduction in the amount of sputum preceding each febrile bout, simply because obstruction to free drainage has occurred. Between times the sputum is yellow or green, invariably profuse, but almost never offensive; for as in chronic bronchiectasis, long-established resistance eliminates the anaerobes. The fingers and toes are often clubbed, and X-rays show a consolidated area of lung, usually in an upper lobe or in the apex of a lower, with one or more smallish cavities discernible in its midst. This appearance may greatly resemble chronic fibro-cavernous tuberculosis or a cavitating neoplasm, but neither acid-fast bacilli nor malignant cells are present in the sputum (unless indeed secondary tuberculous infection has occurred), and bronchoscopy helps to eliminate cancer. This investigation must never be omitted, for it is also necessary to exclude any other organic bronchial obstruction or an impacted foreign body. The sputum yields a mixed growth of organisms amongst which are usually found streptococci, staphylo-

cocci, pneumococci *H influenzae* and sometimes *B proteus* and *B coli*. One must also make sure the infection is not an actinomycotic one.

Bronchography is chiefly of value in outlining the normal lung, for the Lipiodol seldom enters the affected bronchi which are full of pus and debris and it never outlines the cavities within.

Treatment

If a very high level of penicillin is maintained in the blood stream, spill-over takes place from it into abscess cavities that is not achieved with normal dosage. Patients with chronic pulmonary cavitation should therefore be given 2,000 000 units of penicillin a day, and this should be continued for at least a fortnight or three weeks according to the response. Other antibiotics are used if the predominant organisms are penicillin resistant. Postural drainage is always employed and persisted in, but owing to the devious complexity of the abscess system, is never fully effective. Secondary anaemia is corrected, a high fluid intake secured, and the general nutrition cared for.

By these measures, but chiefly with the aid of antibiotics, the infection is brought under control and the amount of sputum greatly reduced. If the patient is too old or ill for pulmonary resection, or the disease is already bilateral, no more can be achieved and he must continue daily postural drainage reserving antibiotics for re-infection and for the more acute recrudescences of the disease. In all others the definitive treatment of choice is excision of the affected segment, lobe or lung.

External drainage of chronic abscess cavities is absolutely contra-indicated and should never be performed.

STAPHYLOCOCCAL ABSCESSSES

It would be difficult to find a more striking contrast than that between staphylococcal and aspiration lung abscesses. As we have seen, the latter depend upon the inhalation of a septic embolus during a period when the cough reflex is in abeyance and therefore are commonly preceded by loss of consciousness or by anaesthesia. They are usually associated with gross dental sepsis. They are never seen in children. They affect particular lung segments. The pus coughed up from them is profuse and generally stinking and yields a mixed growth of organisms. They have comparatively thick, regular walls, being radiologically opaque in their early stages, but later showing a fluid level of pus. When surgical relief is necessary it

must often take the form of primary pulmonary resection, and although they are sometimes complicated by empyema, such an accident is a late development and nowadays unusual.

Staphylococcal abscesses, on the other hand, are usually caused by infection reaching the lung by the blood stream from a staphylococcal focus elsewhere in the body; and neither the cough reflex nor loss of consciousness have anything to do with them. There is no relationship with dental sepsis. They are most common in infancy, but are sometimes seen in debilitated adults; and occur anywhere in the lung, without selection of particular segments. Little or no pus is coughed up from them, but what there is is never offensive and yields a pure growth of *Staphylococcus aureus*. Their walls characteristically resemble soap bubbles, being very thin, cyst-like, and transient (X-ray 24). They are often empty from the time they are first seen; and are commonly complicated by empyema very early in their course. Surgery is seldom required in connection with them, and pulmonary resection never.

The explanation of these remarkable differences probably lies in the mode of origin of the abscesses. As the organisms reach the lung by the blood stream rather than by the bronchi, they are filtered off in the alveolar plexuses and give rise to pneumonia and to obstructive lesions much further distally in the bronchial tree than the comparatively gross emboli aspirated from the nose or mouth. Groups of alveoli are affected rather than whole lung segments, and the alveolar walls break down to form delicate cyst-like abscesses, rapidly expanded by the partially obstructed air flow until they cease altogether to resemble abscesses, but rather the bullae of severe emphysema (X-ray 42). Many burst at once, contaminating the pleural space and causing a pyopneumothorax which may continue to be subjected to the same increasing positive pressure as was the ballooning cyst which preceded it. Such staphylococcal empyemas are exceedingly common in infancy and are always secondary to a staphylococcal pneumonia of this type. Many, if not most, of the 'congenital lung cysts' and examples of 'spontaneous pneumothorax' in infancy are due to the same cause. As a rule they appear, grow, blow up, and disappear all in a day or two, but a few linger on for weeks or even months, and it is possible that some groups of localized bullae seen in young adults whose lungs are otherwise normal are the monuments of a past staphylococcal infection. Such cysts when in the base of the lung are sometimes mistaken for herniation of loops of bowel through the diaphragm, because of their distended walls, transient fluid levels, and rapid change of outline; but the history and course

of the illness soon differentiate the two and if any doubt remains a barium follow through settles the matter

Staphylococcal pneumonia was first recognized during the devastating influenza epidemic which swept the world in 1918-19 and killed many more people than did the preceding war. The pneumonia was its terminal stage, and the cause of death representing a final secondary septicaemia. Patients were prostrated, with high fever and a relatively slow pulse, weak and poor in volume. They had clammy skins and a peculiar pinkish cyanosis which was a feature of the disease. The copious frothy sputum, sometimes blood streaked, grew *Staphylococcus aureus* in profusion and death followed from respiratory insufficiency and profound toxæmia. Although their prognosis has been completely altered by antibiotics such virulent infections are still occasionally seen in influenzal outbreaks but a much milder variety is very common in infancy.

If X rayed in the early invasive stage, the lungs show nothing due to patchy bronchopneumonia which may be diffuse or localized. When adults (who can expectorate) are affected, there is at first no sputum but after a day or two it becomes more plentiful though it is never offensive. Further radiography then shows small multiple cavities in the midst of the areas of consolidation and these rapidly distend until they assume the appearance, not of abscesses but of alveolar cysts which may become so big that they cause mediastinal displacement and embarrass respiration. Should this happen, and there is no speedy improvement with penicillin its relief is more important than any risk of pleural contamination or pneumothorax, and a needle must be inserted into the cyst to relieve the positive pressure in it. Sometimes it is necessary to leave a tension pneumothorax valve *in situ*. Apart from antibiotic therapy the chief problems in treatment are mechanical. A positive pressure is just as apt to build up in the empyema spaces as it is in the cysts themselves, and it is usually air that has to be withdrawn, rather than pus. Indeed it is remarkable how seldom one needs to aspirate pus from such empyemas although initially they may seem to be full of it. Occasionally of course it must be aspirated, and even more occasionally an intercostal catheter has to be inserted but much more often the pus vanishes altogether leaving an air filled space which compresses the underlying lung. I recall a baby who had run the typical course of a staphylococcal pneumonia and had been well for more than six weeks, except that the left lung was collapsed against the mediastinum and seemed to be covered by a thick crust. It was sent into hospital for the lung to be decorticated, but when the houseman inserted a

needle into the pleural space the pressure within blew the plunger out of his syringe. An X-ray taken the following day showed the lung to be completely re-expanded.

No matter how forbidding the chest X-ray appears, the surgeon should not allow his anxious medical colleagues to persuade him to operate upon these children. aspiration of air or pus and the very occasional insertion of a drainage tube are the only surgical measures required. The great majority get better with patience and penicillin.

MISCELLANEOUS CONDITIONS GIVING RISE TO ABSCESSSES

Friedlander's bacillus

Friedlander's bacillus sometimes causes a specific pneumonia with abscess formation. The infection begins as an acute bronchopneumonia which soon becomes confluent, so that a whole lobe may develop gangrene if the process is not arrested by antibiotics. The ragged abscess cavities formed tend to be very large and to contain substantial lung sloughs which can be seen floating in the pus and are likely to impede bronchial drainage. The sputum yields an almost pure culture of *B. friedlanderi* which is insensitive to penicillin but responding to Chloromycetin or aureomycin. If the infection cannot be brought swiftly under control with one of these, primary lobectomy is indicated, especially if a slough is visible in the X-rays.

A chronic form also occurs which radiologically resembles chronic pulmonary tuberculosis, Friedlander's bacillus being present in the sputum but no tubercle bacilli. It also calls for excision.

Actinomyces

Actinomyces live saprophytically in a considerable proportion of healthy lungs, and it is not known upon what factors their pathogenicity depends. Infection may be primary, or secondarily imposed upon some other lesion; and when it occurs is followed by an indolent excavation of the lobe which can easily be mistaken for tuberculosis or chronic suppurative pneumonitis, but is identified by the discovery of the fungus, with its yellow granules, in the sputum. The cavitation may reach the pleura and lead to an empyema, but usually there is so much pleural reaction that the layers are adherent; in which case sinuses break through an intercostal space and discharge actinomycotic pus upon the skin surface.

Treatment consists of massive dosage with penicillin. 2,000,000 units should be given every day for a period of not less than six



25

P. A. view of normal right bronchogram. Note that there is no alveolar filling, that all segments are filled, and that none has been allowed to spill into the opposite lung.



6

lateral normal right bronchogram. Note the 3 segments of upper lobe—apical (with 'Y' and posterior. Below anterior segment is middle lobe bronchus. Posterior to origin of middle lobe is lower lobe and inferiorly anterior, lateral and posterior basal segments of lower lobe.



27

A left bronchogram of X-ray 2, showing gross bronchiectasis in the collapsed left lower lobe. Heavy alveolar filling of the rest of the left lung obscuring the bronchial anatomy.



28

A right lateral bronchogram showing bronchiectasis confined to the middle lobe.



29



30

X-ray 29 Gross cystic bronchiectasis in a shrunken and fibrotic right lung dragging mediastinum towards it. Dense opacity at right apex is a wax plombage performed many years ago in the belief that disease was tuberculosis. Phrenic evulsion made bronchiectasis worse. Pneumonectomy gave excellent result.

X-ray 30 A right lateral bronchogram showing complete filling of the bronchial tree except for the anterior segment of the upper lobe. An early carcinoma obstructed the segmental bronchus. Failure to fill is always significant provided bronchographic technique is good.



31



32

X-ray 31 A lateral view of the sequestered pulmonary segment seen in X-ray 35. Its posterior position in the chest is characteristic.

X-ray 32 A right bronchogram proves the cystic area seen in X-rays 31 and 35 to lie wholly outside the normal bronchial anatomy.

weeks, and in any case maintained for some time after all trace of the infection has been extinguished.

Infected cysts

Infected cysts, whether congenital or acquired, behave like chronic abscesses, and are sometimes very difficult to distinguish from them as their own character is obscured. No matter how efficient the drainage from them, they will of course never diminish in size because their walls are rigid and epithelialized.

Congenital bronchogenic cysts

Congenital bronchogenic cysts, if they communicate with a fairly large bronchus, inevitably become infected, and the mucus they secrete is coughed up as pus. They must be excised, and it is easier and better to do this before they become infected than afterwards (X rays 33-34)

Mediastinal dermoids

Mediastinal dermoids are peculiarly prone to blood borne secondary infection and subsequent fistula formation, and the same considerations apply to them.

Sequestered lobes

Sequestered lobes often contain one or more abscess cavities which establish fistulous communications with adjacent normal tissue so that pus is coughed up from them. They are mistaken for ordinary lung abscesses and for empyemas and sometimes tubes are inserted into them. Being essentially a mass of aberrant tissue they are unaffected by drainage and require to be excised *en masse* (X rays 31-32)

Hydatids

Hydatids, once they rupture into a bronchus get infected and, if scolices or daughter cysts are not found in the sputum pass unrecognized. History of residence in areas where the disease is endemic is suggestive and the presence of other cysts or of a positive Casoni reaction, conclusive. When a solitary cyst is infected and dies, however its identity is unlikely to be established before its removal (X rays 43-44)

BRONCHIECTASIS

BRONCHIECTASIS is one of the commonest diseases of the lung and is met with in patients of all ages, but it is most characteristically seen in children and young adults. It is essentially a chronic disease, and though its clinical manifestations are sometimes not apparent until well on into the patient's maturity, by far the greater number of cases have their origin in infancy. In later life its symptoms are overshadowed by those of the chronic bronchitis and emphysema which complicate its long presence.

Aetiology

When a bronchus is obstructed from any cause the mucus secreted by glands beyond the blockage is dammed up in the collapsed lobe or segment, and passively distends the bronchi. If the obstruction be relieved, the mucus escapes, the bronchi return to normal, and no harm is done; but if those pent-up secretions become infected, the pool of mucus turns to a pool of pus, the mucosa is ulcerated, the submucosal tissues are inflamed, with accompanying lymphocytic infiltration and fibrosis, and the bronchial wall is weakened, eroded and distorted. Such damage is permanent; and once it is established it can be cured only by surgical excision. All other means of treatment are purely palliative. Clinical bronchiectasis, therefore, depends upon two factors: (1) *Collapse*

(2) *Infection*

In the adult, pulmonary collapse is generally due to intrabronchial obstruction, for example a neoplasm, or a plug of tenacious secretion; but in the infant, whose bronchial walls are soft and pliable, the cause is usually pressure upon them from without, i.e. from enlarged lymphatic peribronchial and hilar glands. By far the commonest source of such enlarged glands is the *primary tuberculous complex* which at some time or other affects 80 per cent of urban children, and the lymphatic glands are larger and firmer during this infection than at any other time. Enlargement also occurs, however, during *whooping cough* and the pulmonary complications of *measles*; so it is not surprising that collapse of parts of the infant lung are common. Because of the anatomical arrangement of the glands

AETIOLOGY

round the bronchi the parts affected most often are the lower l and lingular segment on the left side, the middle lobe upon the r Usually these re-expand and the mucus escapes from them when glands decrease in size but in some infection occurs and bronchi tasis results

Although it is true that the cause of bronchiectasis is most o collapse and infection of a lung segment during infancy chr bronchial obstruction at any time results in the same damage. Ht the urgency in relieving postoperative atelectasis by prompt bronc scopy and in maintaining antibiotic cover so long as it persists in extracting aspirated foreign bodies, which inevitably com obstruction with infection, as quickly as possible. Pulmonary i puration and chronic tuberculosis also give rise to secondary b chiectasis if as is commonly the case, the bronchi of the infla areas are at some time occluded during the disease and since advent of effective chemotherapy the surgical treatment of aspira lung abscess and suppurative pneumonitis is often confined to d ing with the bronchiectasis they leave behind them. Under ti circumstances any segment of the lung may be involved and if piration for example played an important part in the aetiology bronchiectasis one would expect its distribution to be the sam that of aspiration abscesses. The postero-lateral segments of upper, and the apical parts of the lower lobes are indeed pl where localized areas of post inflammatory bronchiectasis are fou but it is significant, and confirmatory of the predominantly infla origin of the disease, that in the great majority of patients it is left lower lobe (X ray 27) the lingula, and the middle lobe (X ray in that order which are most frequently and severely involved. these parts between them account for 75 per cent. of the incide of the disease. Curiously enough the apical segment of the left lc lobe is quite often spared, while its basal segments and the lingula affected together or in combination with the middle lobe on opposite side. It is also common to find the middle lobe alone eased, as it is so susceptible to glandular compression. The r lower lobe ranks a poor fourth to these three areas, followed by remainder of the upper lobes. Sometimes bronchiectasis is patc distributed throughout both lungs, rendering surgery quite im sible but at others the whole of one lung is damaged whereas opposite side is quite unharmed, and pneumonectomy can the carried out.

Diagnosis

Patients are most often seen when they are children or young adults. If children, they are nearly always mouth breathers, and often undersized and apathetic. They tend to swallow their sputum rather than expectorate it, and some vomit in consequence. Adults may appear sallow and toxaemic. Both describe their cough, which is loose and productive of yellow or greenish, but non-offensive, sputum, as having existed for as long as they can recall, or since childhood. It is worse when they first rise in the morning, and is sometimes aggravated by bending down at work. In some constant expectoration of pus can render work difficult, society an embarrassment, and life a burden; but in most infection is by no means constant, and the purulent sputum only intermittent. This is because natural resistance, or chemotherapy, may extinguish it for considerable periods, until it recurs after a cold in the head, tonsillitis, or pus from infected nasal sinuses aspirated into the bronchi. This latter process becomes a vicious circle, and is particularly common at an age when septic adenoids and tonsils provide a source of infection for the lung. Pus is coughed up to infect the nasal sinuses, from which it drips down again to re-infect the lung. As a result most young people with bronchiectasis also have nasal obstruction or deformity, polypi, septal deviation, or chronic antritis; and it is futile to treat one without the other.

The history probably includes acute febrile attacks of varying frequency, recurrent pneumonia, pleurisy, or even empyema. Remissions, during which there is little or no sputum, are usual but not invariable. During them the excoriated bronchial wall may bleed, so that the patient spits up blood. In a young adult this is very apt to raise suspicions of tuberculosis, even though tubercle bacilli are never discovered in the sputum, and the results can be tragic for the patient. Two patients in particular come to my mind: the first was a farm labourer in his late thirties. He had coughed up a little blood fifteen years previously and was at once admitted to a sanatorium where the usual tests were done with negative results. His chest X-ray showed some mottled shadowing in the right lung. Bronchograms would have revealed this to be due to bronchiectasis, but they were never done, and his phrenic nerve was crushed instead. Permanent paralysis of his diaphragm resulted, so that he could not cough up the pus from his right lung and the X-ray shadowing got worse. An extrapleural pneumothorax was next carried out, and the space so formed filled with paraffin wax to convert it into a wax plombage.

(X ray 29) Small fragments of wax later ulcerated into his lung and were coughed up at intervals during the next ten years. Finally a lateral thoracoplasty was performed. None of these measures of course made any difference to the patient's symptoms, except the phrenic crush which made them much worse. Eventually a right pneumonectomy rid him of his bronchiectasis, wax plombage and all.

The other case is that of a woman of 42. At 18 she, too, had had an haemoptysis and been promptly shut up in a sanatorium. In spite of consistently negative sputum tests she was kept there for two years before being released to rest at home. She rebelled, got married, and became pregnant. Her medical advisers promptly terminated the pregnancy and had her sterilized. As a result her husband left her, her health became much worse and she was readmitted to another sanatorium where she also remained for several years, her sputum ever increasing in amount, and her left lung slowly shrinking into a fibrotic bag of pus. A bronchogram was never performed. She, too, was cured by left pneumonectomy but not before much bronchitis and bronchospasm in the opposite lung had made surgery desperately hazardous and twenty years too late to prevent her youth, her child and her husband being sacrificed to a misdiagnosis.

This alternation of haemoptysis with purulent expectoration is the basis of the old-fashioned and quite artificial division of the disease into 'wet and dry' varieties, these phases merely marking remissions and recurrences of renewed infection but in many patients the cough is consistently productive and haemoptysis not seen at all. If the disease is severe the fingers and toes may be clubbed.

Bronchiectasis is recognized clinically by its history and symptoms but it can be diagnosed only by bronchography for surgical diagnosis means more than simple recognition. It implies accurate definition of the extent of the disease of each segment that is involved, and of every segment that is not. Only then can a precise assessment be made and operability determined.

Physical examination of the chest perhaps reveals moist added sounds at the left base which are more evident after coughing or suggests an area of collapse but quite often it is wholly negative, and the diagnosis must never depend upon it. Similarly plain X rays sometimes show mottling due to accumulated pus in the affected area, or cyst like cavities or even collapse of a lobe or segment but they may be altogether inconclusive, and a normal radiograph of the chest never excludes bronchiectasis. It is important to make sure tubercle bacilli are not present in the sputum and to culture and determine the antibiotic sensitivities of those organisms which are. If there

is any possibility of a foreign body or an organic stricture being present bronchoscopy must be carried out. The conclusive and indispensable step in the diagnosis of bronchiectasis is bronchography; and this is indicated whenever a patient's history suggests the disease, whether physical signs are wanting, or the chest X-ray seems normal, or not.

Bronchography

Bronchograms demand a high technical standard if they are to be of any value, and inadequate ones are seriously misleading and should always be rejected or repeated. Their performance calls for care and skill, and for co-operation from the patient and the radiographer, but the technique is easily learnt and should be a common routine in every chest clinic. They are obtained by instilling iodized oil into the trachea so that complete radiographic definition is obtained of all the bronchial segments of both lungs. It is just as important to outline the normal as the abnormal, and no opinion ought to be given or plans for treatment made if this is not achieved (X-rays 25 and 26).

In children under 10 or 12 general anaesthesia is usually necessary and the child should be kept in hospital for at least one night; but bronchography is carried out on adults under local anaesthesia in the out-patient department. In both it is important to ensure a period of postural drainage beforehand so that pus does not interfere with complete filling of the bronchi, and coughing during the procedure is diminished. The patient should also have fasted for the preceding four or five hours. It is most important to gain the patient's complete confidence and co-operation, allaying nervousness, and explaining exactly what is required. If this is done effectively one can often perform bronchography on quite small children under local anaesthesia.

Certain technical points must always be observed:

- (1) One side is filled at a time, the right side should always be done first, so that P-A and true lateral X-rays can be obtained. Only when these are seen to be satisfactory is the left side proceeded with, a left oblique view being taken instead of a lateral to avoid confusion with the right bronchial tree.
- (2) No oil is allowed to spill over into the opposite lung where it will be superimposed and confuse the lateral view. For this reason the patient is kept tilted to the side which is to be

examined, and must not cough during the process. Oil coughed up and swallowed or spilled over also reduces the quantity available for planned filling and poor films result.

- (3) Alveolar filling which obscures the bronchial definition must be avoided. This is achieved by using a fairly viscous medium and by not wasting time between the instillation of the oil and the taking of pictures. The X ray plates should be in position ready for exposure before bronchography begins and the patient is rehearsed in positioning.
- (4) As has already been emphasized, filling must be complete and embrace all segments. After each side is done the wet films are examined and if a segment is found inadequately filled more oil is injected with the patient in the appropriate posture to fill it (X ray 30).

Many techniques are employed for bronchography in adults and the individual should use whichever method gives the best results in his hands. After trying most of them over a period of years I have come to prefer the following which is easy to learn, not unpleasant for the patient, and permits complete bronchograms of both lungs to be obtained at one sitting, and indeed bronchoscopy also if required.

The patient lets a 50-mg. Nupercaine tablet, given 20 to 30 minutes beforehand dissolve on the back of his tongue. The pharynx and trachea are then treated in exactly the same way as for a bronchoscopy. The operator sits facing the patient and he is told to breathe quietly in and out through the mouth. With a swab the tongue is drawn gently forwards and a fine gum-elastic catheter on a curved introducer passed over it, and down through the glottis to enter the trachea. The introducer is withdrawn and the position of the catheter confirmed by asking the patient to phonate or to close his lips and breathe through the tube. If difficulty is encountered in passing the catheter the index of the gloved left hand may be used to hook the base of the tongue forward, palpate the epiglottis and hold it forward while the catheter is guided down between the cords. If preferred, the catheter or even a Ryle's tube can be passed through the nose.

The patient now sits on the tiltable X ray table with the operator's left arm round his shoulders leaning back a little, and inclined to the right, so that he is supported. The injection of oil, and the changes of posture which follow are conducted in one smooth succession of movements, the patient continuing to breathe steadily

is any possibility of a foreign body or an organic stricture being present bronchoscopy must be carried out. The conclusive and indispensable step in the diagnosis of bronchiectasis is bronchography; and this is indicated whenever a patient's history suggests the disease, whether physical signs are wanting, or the chest X-ray seems normal, or not.

Bronchography

Bronchograms demand a high technical standard if they are to be of any value, and inadequate ones are seriously misleading and should always be rejected or repeated. Their performance calls for care and skill, and for co-operation from the patient and the radiographer, but the technique is easily learnt and should be a common routine in every chest clinic. They are obtained by instilling iodized oil into the trachea so that complete radiographic definition is obtained of all the bronchial segments of both lungs. It is just as important to outline the normal as the abnormal, and no opinion ought to be given or plans for treatment made if this is not achieved (X-rays 25 and 26).

In children under 10 or 12 general anaesthesia is usually necessary and the child should be kept in hospital for at least one night, but bronchography is carried out on adults under local anaesthesia in the out-patient department. In both it is important to ensure a period of postural drainage beforehand so that pus does not interfere with complete filling of the bronchi, and coughing during the procedure is diminished. The patient should also have fasted for the preceding four or five hours. It is most important to gain the patient's complete confidence and co-operation, allaying nervousness, and explaining exactly what is required. If this is done effectively one can often perform bronchography on quite small children under local anaesthesia.

Certain technical points must always be observed

- (1) One side is filled at a time, the right side should always be done first, so that P-A and true lateral X-rays can be obtained. Only when these are seen to be satisfactory is the left side proceeded with, a left oblique view being taken instead of a lateral to avoid confusion with the right bronchial tree.
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through his mouth the while and suppressing any impulse to cough.

A 20-cc syringe containing 15 cc of Neo-Hydriol to which 5 G. of sulphadiazine has been added is attached to the catheter, and 5 cc. injected to fill the posterior portions of the lower lobe. The patient then brings his own left arm across to grasp the right border of the table so that he leans well forward and to the right, and a further 5 cc fills the middle lobe and anterior parts of the lower, finally the remainder is quickly instilled, and the patient lies for a moment or two on his right side while the foot of the table is elevated. This ensures filling of the upper lobe bronchus, and the X-rays are taken forthwith. When the wet plates have been inspected and satisfactory filling of all segments of the right lung confirmed, exactly the same proceeding is carried out on the left, the patient leaning this time to that side, bringing his right hand across to grasp the left border of the table, and so on.

Bronchograms on small children under general anaesthetic are not wholly devoid of danger and it is essential that the anaesthetic is given skilfully and after full preparation. To obtain good pictures respiration must be as quiet and regular as possible, and whatever pus is in the bronchial tree should be tipped, or sucked, out prior to bronchography. After an intratracheal tube has been inserted and the breathing has become steady, the child is held in the same series of postures as have already been described while the oil is injected directly through the rubber of the intratracheal tube. The amount used depends of course upon the age and size of the child, but is about 1 cc per year of age. After films of the right side are completed the procedure is repeated on the left; and finally the whole bronchial tree is aspirated clear with a gum-elastic catheter passed down the intratracheal tube. The child should then be nursed head-down until consciousness and coughing are restored.

The type of radio-opaque oil and the technique of bronchography employed is largely a matter of personal preference. The mixture of Lipiodol, or Neo-Hydriol, and sulphadiazine described has the advantage of giving excellent definition without alveolar filling. It is somewhat viscous and consequently a little difficult to inject; and is not so readily cleared from the lungs as is Dionosil, which should be used if it is important to avoid residual opacities, but does not give such good definition. The oily preparation of Dionosil compromises between the two and is used for the investigation of patients with tuberculous bronchiectasis.

As an alternative to the use of an oral or nasal intratracheal catheter,

oil may be injected directly through the cricothyroid membrane with a short guarded needle. The patient lies on his back with his neck extended over a small sand bag. A skin bleb of local anaesthesia is raised over the cricothyroid membrane but it is quite unnecessary to inject any anaesthetic solution into the trachea as the iodized oil is non-irritant. The needle of the Lipiodol syringe is then passed through the bleb into the tracheal lumen, while the larynx is steadied between the fingers of the left hand. The position of the needle is checked by the withdrawal of air into the syringe, the patient sits upright, and injection and positioning is carried out as already described. After withdrawal of the needle the skin is gently pinched and massaged round the puncture so that no sinus remains. The method has the disadvantage of not being suitable for bilateral bronchography but avoids the necessity for pharyngeal anaesthesia.

Treatment

Only when bronchograms are complete can treatment be planned. Whenever disease is sufficiently localized and the remainder of the lung healthy surgery is indicated, for it holds out the only hope of cure. Quite apart from the hazards of repeated pneumonia, of pleurisy of empyema, and the misery of constant expectoration of pus the longer bronchiectasis remains the more likely is damage to the other part of the lung from aspiration from associated bronchitis and from emphysema. For this reason extirpation of a bronchiectatic lobe in elderly patients is often disappointing, for much of their disability cough and sputum stems not from the bronchiectatic part but from the rest. Furthermore it must never be forgotten that a patient with pus in his lung has constantly hanging over his head, like a sword of Damocles, the danger of a metastatic cerebral abscess and if suppuration is extensive of amyloid disease. The sooner it is removed, therefore the better and there is no justification for conservative treatment in a young adult in whom resection is feasible. In children surgery is better postponed until they are big enough to co-operate during the postoperative phase unless of course the disease is obviously interfering with health and growth. In patients over 45 very careful assessment is necessary of the state of the remainder of the lung, for they may be unable to tolerate the loss even of a partially diseased lobe and their symptoms may not in any case be relieved. But when a lobe is found to be wholly collapsed, as is quite often the case, nothing but good can come from its removal. Not only is the patient rid of a functionless bag of pus but the unoxxygenated blood which entered it through its pulmonary artery is

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diverted elsewhere in the lung to take up oxygen, and an actual decrease of breathlessness must result. When a whole lung is destroyed in such older patients, or severely damaged, great benefit sometimes follows pneumonectomy, and contra-lateral bronchospasm, largely reflex in character, is relieved.

In an otherwise healthy subject the maximum lung resection tolerated is pneumonectomy upon one side when the opposite lung is healthy; or removal of both lower lobes with the lingula or middle upon one side, or the equivalent of this tissue in some other distribution. The question, however, which must occupy the surgeon is not 'How much can I take away?' but 'How much can I leave behind?' If, for example, bronchograms show three basal segments of a lower lobe to be diseased, but the apical segment normal, it is unjustifiable to perform lobectomy. The basal segments alone should be taken, and the apical spared. When disease is bilateral, as it very often is, due to involvement of lingula and middle, or left lower with lingula and middle lobes, the worst side should be resected first, and the patient warned that though his sputum will be less it will not be abolished, and a second operation will be necessary in three months' time.

The degree of bronchial distortion differs widely from mild 'tubular' bronchiectasis to the gross cystic type in which the dilatations may contain fluid levels and resemble a chain of lung abscesses, which in a sense they are. The decision to operate in milder cases rests upon the degree of disability suffered by the patient. How much sputum does he cough up in a day? Does he ever spit blood? How many days are lost each year from work, or from school, because of febrile chest illnesses, or pleurisy, or 'bronchitis'? Upon the answer to such questions depends the decision to operate, and as I have already said, they must be weighed with particular gravity if the patient is past middle age.

Operation

As soon as the decision to operate has been made, and the exact extent of resection necessary determined, preparation of the patient begins. Surgery need never be hurried, and the optimum time both as regards the patient's condition and the weather should be selected, for many bronchiectatics enjoy much better health during the spring and summer than in the winter months. In children and in those in whom it has not already been dealt with, upper respiratory tract disease must be corrected as far as possible. Infected tonsils and adenoids or nasal polypi are removed and the cranial sinuses and

antra inspected and treated as required. It is often necessary to admit children to hospital for these things to be done first, and then, after they have had a holiday by the sea, to re-admit them for lung surgery.

In hospital postural drainage of the affected segments is begun at once, and should be carried out at least twice daily for periods of about twenty minutes at a time but preferably for as long and as often as the patient will tolerate it and pus drains. The use of a Nelson bed is convenient (but not essential) for this purpose the position of the body being adapted to the situation of the affected lung segments. For example if either of the lower lobes are involved the patient lies face downwards, with head and shoulders well below the level of the hips if lingula or middle lobes, then supine the right upper lobe drains best if the patient lies on his left side and so on. Reference to good lateral bronchograms is necessary when planning the suitable position. During posture forced coughing helps drainage as does also percussion on the chest wall by the physio-therapist. Aerosol inhalations of penicillin are used to help subdue infection and diminish sputum. If penicillin resistant organisms predominate other antibiotics are used. When sputum has been reduced to a minimum the patient is ready for surgery.

The operation which usually involves the removal of several segments or a lobe is planned to extirpate all the diseased bronchi and to preserve all the normal lung. If the bronchiectasis is confined to one side the operation is performed with the patient in a lateral position providing that the pus-containing lung is sealed off by an endobronchial blocker and efficient aspiration is maintained but when it is bilateral it is safer to have the patient lying face downwards on the table so that all secretions drain naturally towards the mouth and there is no risk of spill-over to dependent parts of the lung. This position is also used routinely for children into whose small bronchi a blocker can not in any case be passed. At the end of the operation underwater-sealed drainage tubes are always left in the pleural cavity to remove air and effusion and to promote the early re-expansion of the residual lobe. These drains can generally be removed after 24 or 48 hours.

By far the commonest postoperative complication is collapse of the remaining lobe or segments on the side of the operation—the upper lobe when the lower and the lingular segment have been removed (X ray 11) and, very frequently the apical segment when it alone has been spared and the remainder of the lower lobe excised. Atelectasis of this kind may occur immediately but is more frequent

about the third or fourth postoperative days when there is oedema near the healing bronchial stump, which is closely related to the maining airway. Its incidence is also much affected by the patient's age and co-operation, being greater in children and teenage girls than in intelligent adults and those who make an early effort to cough even though the act be painful. Every effort is made to minimize the pain by judicious use of analgesics without suppressing the cough reflex itself. The patient is encouraged to sit out of bed on the day following operation, and soon to take active exercise. From the time that consciousness is regained after operation the patient carries out the breathing exercises taught preoperatively and is helped to cough at regular intervals. Postural drainage is also helpful in aiding expansion by gravity. Dehydration makes the sputum more tenacious and difficult to cough up, so it is important that the patient should never be thirsty and that his tongue remains moist.

Lobar collapse is marked by a sensation of tightness across the chest, dyspnoea, and a rise of pulse rate and temperature. The trachea or apex beat is displaced to the same side as the airless lobe; the percussion note is impaired over it, and breath sounds are absent or bronchial in character. The diagnosis is confirmed radiographically, when the area in question is seen to be opaque and the mediastinum drawn towards it.

Each hour that elapses between collapse and its relief by bronchoscopic aspiration makes that relief more difficult to achieve. It is often the case that the mucous plug responsible cannot be sucked out but must be pulled from its lodgement with grasping forceps. If the first attempt to re-aerate the lobe is not successful the collapsed lung must be protected from danger of secondary infection (and hence from further bronchiectasis) by systemic penicillin in full dosage. Postural drainage is continued, and bronchoscopy repeated until such time as re-expansion occurs. Thereafter residual effusion is aspirated, and chest mobility and full aeration of the lung promoted by vigorous exercises and early activity. Effusions should never be aspirated in the presence of atelectatic lung, for to do so increases negative pressure in the pleural cavity tending to drag the bronchial obstruction farther and farther out into the small bronchi, so making its removal more difficult. Lobes become wholly airless, not because they are compressed by effusions, but because the airway to them is blocked.

Should the sputum continue to be purulent after an operation for bronchiectasis, and no source of pus remains in the nasal sinuses, it generally means: (1) that further disease exists in segments left

behind (2) secondary bronchiectasis has occurred in a residual segment postoperatively collapsed or (3) the formation of a puddle in the bronchial stump if this has not been divided flush with the remaining stem bronchus. Any of these causes calls for further bronchograms and possibly resection of the areas involved, or in the case of the stump, re-amputation.

Palliative treatment

As already described the palliative treatment of those unsuitable for surgery lies in regular and accurately planned postural drainage of the affected lung segments, acute exacerbations of infection being subdued by antibiotic therapy. Every effort should also be made to treat upper respiratory tract infection and if present, nasal deformity or obstruction.

CYSTS OF THE LUNG

LUNG cysts comprise a complex and diverse group of disorders, congenital or acquired, single or multiple, some due to infections or infestations, some to degenerative conditions, some of unknown aetiology. Pathological changes in the lung soon after birth may be mistaken for true congenital lesions, and defects first seen in later life may have their origins in developmental faults, but generally speaking congenital cysts are bronchogenic, have true walls and a lining mucosa, contain mucus, and produce symptoms after secondary infection, while those that are acquired are mostly pseudo-cysts created by the breaking down of alveolar walls to form confluent cavities having no true wall of their own and no mucosal lining, containing no fluid, and producing symptoms due to their mechanical enlargement or actual rupture. Hydatid infestation is an exception and forms a special case which will be considered separately.

When empty, a congenital bronchial cyst, an emphysematous bulla, a staphylococcal abscess, a tuberculous tension cavity, and an intrathoracic portion of stomach or loop of bowel can produce remarkably similar appearances on a plain X-ray film. but the symptoms that accompany them, their clinical course, and specific investigations (such as the finding of tubercle bacilli or the taking of a barium meal), usually serve to distinguish them. When full of fluid a cyst, being opaque to X-rays, exactly resembles an ovoid or spherical solid tumour, and may even be mistaken for an encysted effusion or an empyema.

CONGENITAL CYSTS

BRONCHOGENIC CYSTS

These cysts are in all cases characterized by a lining of respiratory epithelium, cuboid or columnar, with mucous secretory glands, and by a wall in which bronchial elements, fibrous and elastic tissue, and fragments of cartilage, are present in disorderly array. It is the possession of these features which principally distinguishes them from all other cysts of the lung and help to determine their behaviour. Kindred anomalies such as conjoined and supernumerary ribs, but particularly aberrant pulmonary vessels and unusual segmental fissures, sometimes occur and are strong evidence of congenital origin. The right lung is more commonly affected than the left.

As the developing lung buds grow out from the primitive foregut of the embryo they may be disordered at any point along their course giving rise to a solitary cyst if this occurs proximally, but, if the disturbance is peripheral and many of the branching outgrowths are involved, sometimes to a completely polycystic lobe or lung. If isolated, the cysts contain mucus secreted by the mucosa, and this gradually distends them until they reach a state of equilibrium but if a communication exists between cyst and bronchus, some or all of the mucus drains out, and they will then also contain air and be subjected to changes in its pressure. The more proximal a cyst is the more likely is it to contain mucus, the more distal, air and therefore to communicate with the rest of the bronchial tree (X rays 33 34) The most proximal cysts are always solitary central mediastinal globes, indistinguishable radiologically (as they are full of mucus and consequently opaque) from any other mediastinal tumours in a similar position. They are closely related to the bifurcation of the trachea, and often attached to the carina by a stalk and their surgical importance is primarily one of differential diagnosis.

A little more distally a cyst, or group of cysts may occur in the substance of the lung itself. These quite commonly have a bronchial communication and are therefore not wholly opaque, but contain a fluid level of mucus, or are even quite empty depending upon the position of the orifice in the cyst wall. Providing a cyst opens into a bronchus it is exposed to secondary infection and this is more likely to occur if the cyst is proximally placed, and less likely if it is distal. Once this happens the mucus becomes pus and the cyst behaves henceforward like an epithelialized lung abscess though its wall prevents infection spreading to the surrounding lung, and as a result the sharp definition of the wall is never lost, and no inflammatory reaction is visible outside it. The patient suffers from chronic ill health, has recurrent fevers, and coughs up constant small amounts of purulent sputum and sometimes blood. Cysts of this type, even when symptomless, are often mistaken for tuberculous cavities, and the danger is much greater when they are associated with cough, purulent sputum and haemoptysis but the remarkably fine and regular wall visible above the level of fluid the neatly rounded outline the absence of any related disease or of tubercle bacilli in the sputum, should prevent confusion.

The discovery of a fluid level in the chest may also lead to a diagnosis of lung abscess or of empyema being made. Ill judged attempts at aspiration are likely to contaminate the pleura and cause

a real empyema; and large parietal fluid-containing cysts, even when not infected, have been mistaken for empyemas, and drained by rib-resection. Such drainage of course does not result in a progressive diminution of the cavity, and a long period of illness with a persistently draining sinus continues until it is realized that a cyst is present, and must be excised.

If developmental disturbances take place more distally still, in the ultimate twigs of the bronchial tree, polycystic lobes or whole lungs result, a multitude of small cysts being present with virtually no lung parenchyma at all. It is this condition which is so often mistaken for 'congenital' bronchiectasis; but bronchiectasis is always an acquired condition, even if it is acquired early in life. In congenital cystic diseases the cysts are much bigger and more uniform than is the case of chronic bronchiectasis, the usual distribution of bronchiectasis is not seen, and other stigmata of congenital disturbance are often present, for instance in Kartagena's syndrome when a polycystic right upper lobe is combined with dextrocardia. Just as do single cysts, so may polycystic areas become infected, and give rise to all the symptoms of chronic bronchiectasis; but, as with infected single cysts, there is the same absence of peribronchial inflammation or spread outside the cyst walls. Toxaemia may be severe, marked clubbing of the fingers and toes is common, and the risk of cerebral abscess ever present. Congenital polycystic disease must also be distinguished from the honeycombed lungs seen in the obscure group of xanthomatoses, Letterer-Siwe's disease, Hand-Schüller-Christian syndrome, and eosinophilic granuloma, in all of which there may be honeycombing, and whose chief importance is the frequency with which spontaneous pneumothorax, often bilateral, occurs. The honeycombing is, however, much finer and is always generalized and symmetrical; whereas congenital polycystic disease is usually confined to one lobe, or at most one side.

Congenital cysts, single or multiple, that are not infected are likely to remain undetected except by routine radiography. Because they have real walls they do not distend progressively when subjected to positive pressures, as alveolar cysts or the closely related staphylococcal cyst-abscess may do (X-ray 42), and most of the 'congenital' ballooning cysts described as occurring in early infancy belong to one or other of the two latter groups. Children with congenital cysts sometimes have asymmetrical chests, are undersized, show early clubbing, or are vaguely 'asthmatic'. Haemoptysis, even in the absence of infection, may be a symptom, is more common when the lungs are polycystic, and can be severe.



33

X-ray 33. A congenital bronchogenic cyst situated in the anterior segment of the upper lobe of the left lung. Fluid level of mucus and pus therefore communicate with the bronchus, so it is probably all one cavity. Note however its very fine regular wall.



34

X-ray 34. Bronchogenic cyst in the anterior segment of left upper lobe (lateral view)



35

X-ray 35. Cavity with fluid level situated posteriorly at base of a child's chest. Pus was coughed up by the child. The cavity was drained years previously on mistaken diagnosis of empyema. It is an infected sequestered pulmonary segment. (See also X-rays 31 and 32.)



36

X-ray 36. A child's chest showing a large, dark, well-defined mass in the lower right lung field, similar to X-ray 35.



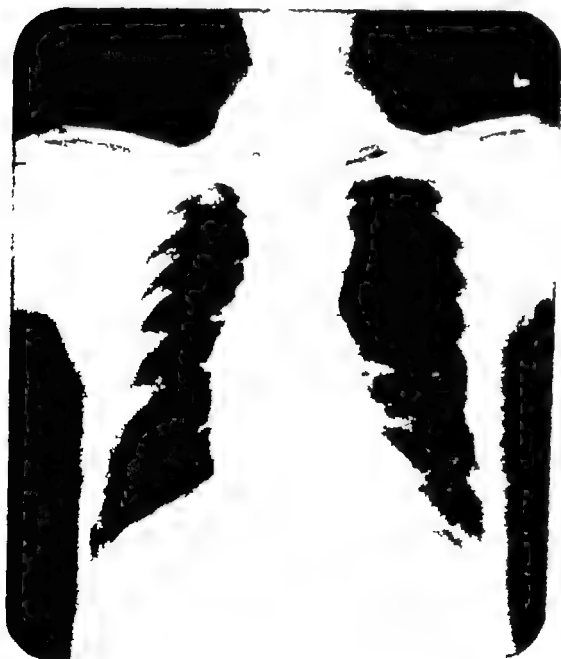
37



38

X-ray 37 Enormous bulla at apex of right lung Left apex was also emphysematous, and left spontaneous pneumothorax occurred twice Opacity seen here developed later, partially cleared after antibiotics Bronchoscopy reveals growth in left upper lobe bronchus DXT made lobectomy possible (See X-ray 38)

X-ray 38 Removal of growth seen in X-ray 37 was impossible until bullae in right lung were excised Afterward lung leaked so much that tension pneumothorax developed (see X-ray 14) Suction produced complete re-expansion Growth on left was removed by upper lobectomy Lateral thoracoplasty prevented lower lobe emphysema



39



40

X-ray 39 An emphysematous bulla at the left apex compressing the rest of the lung and rendering the patient breathless to work For appearances after excision see X-ray 40

X-ray 40 The bulla seen in X-ray 39 has been excised and the lung re-expanded This completely relieved the patient's breathlessness and he has worked comfortably ever since

In later life breathlessness is more prominent, and may be disproportionate to the size of a solitary cyst, especially if the cyst fluctuates in size with respiration. Polycystic lobes are functionless, and dyspnoea is therefore always made less by their resection.

All bronchogenic cysts should be removed if possible for if they are full of mucus they are indistinguishable from tumours, and if they are not they are likely to become infected. They can often be shelled out of the surrounding parenchyma, and any bronchial communications carefully closed afterwards but if they have been long infected segmental resection is indicated. Anomalous vessels add a little to operative difficulties, but if their presence is anticipated risk is much diminished. In the case of a polycystic lobe or lung, lobectomy or pneumonectomy is necessary.

THE SEQUESTERED PULMONARY SEGMENT

This has much in common with the foregoing. During development an outgrowing lung bud becomes wholly divorced from the rest of the bronchial tree and acquires a collateral blood supply of its own. The result is an airless island of pulmonary tissue usually lying posteriorly at the base of the normal lung, but sometimes found elsewhere with no normal bronchial or pulmonary vascular connections at all. It is often cystic, and secondary infection leads to the establishment of fistulous communications (through the base of the lobe to which it is related) with the bronchial tree (X rays 31, 32, 35). Pus is then persistently coughed up and a fluid level appears in the midst of the previously opaque mass at the base of the lung. The condition is rather rare but not very rare; it should be considered in the presence of a persistent basal abscess, with disproportionately little constitutional disturbance and which bronchography demonstrates to lie outside the normal bronchial anatomy. Failure to recognize it leads to such cases as the following.

I. A child of 7 was referred for treatment of recurrent empyema. She had been repeatedly in hospitals since at the age of 4 she had had fever, pain in the back and purulent sputum. X rays showed a posterior basal fluid level, empyema with bronchial fistula was diagnosed, and a drainage tube was inserted. This cured the fever and expectoration for a time, but continued to drain. On its eventual removal she again had pleurisy, a return of all her symptoms, and the X ray appearances were exactly as before (X ray 35). In all, the tube was re-inserted three times in three years with no result. A sequestered segment was excised and recovery was complete.

2. A man of 40 was invalided from the army after an operation for varicose veins. Routine radiography at the time showed a left basal fluid level and a diagnosis of chronic lung abscess following septic infarction was made. This was unaffected by antibiotics, and he was awarded an 80 per cent. disability pension which, to the best of my knowledge, he is still enjoying. The fluid level was in fact in the midst of a sequestered segment which was removed.

At operation the sequestered lung is found lying collapsed and densely adherent both to the base of the normal lung and to the parietes. From the former it can be cut without fear, for no major vessels or bronchi pass between them; but in freeing it from the paravertebral gutter great care is necessary, for here enters its main blood supply, a large aberrant artery rising from the abdominal aorta, and piercing the diaphragm to reach the segment.

ACQUIRED CYSTS

With the exception of hydatids, and the honeycombing of the rare xanthomatoses, the cysts of this category are pseudo-cysts, for they lack regular walls of their own and are outlined only by the tissue they compress. They arise not from the bronchi, but from the alveolar part of the lung, they are much commoner than bronchogenic cysts; they always contain air; and they produce symptoms not from infection but from distention.

ALVEOLAR CYSTS: BLEBS AND BULLAE

The parenchyma of the lung is an elastic web subjected not only to the pressures of the bronchial air stream which ebb and flow from negative to positive with respiration, and fluctuate more violently with coughing; but also to the centrifugal negative suction of the pleural space. Under physiological conditions it is impossible to develop sufficient intrapulmonary tension to rupture a normal alveolus. If, however, an artificial deficiency is produced, a rounded cavity appears and grows disproportionately to the pressure exerted upon it. Its 'wall' consists solely of the neighbouring tissue compressed before it. This, be it noted, is in a lung whose tissues are normal, and not weakened by age or by disease, and the pressures applied are only within the range of natural physiology. The tendency for a cavity to enlarge is greater still if the adjacent alveolar walls are already defective, and readily break down; and much greater if exceptional circumstances raise the pressure within the expanding cyst.

beyond the normal range of respiration. Many factors lead to weakening or destruction of the alveolar walls—suppuration, tuberculosis, or, most commonly of all, chronic degenerative emphysema, in which there is a generalized loss of pulmonary elastic tissue, but a potent force superadded to these is that of a check valve mechanism (X-ray 36)

If the lumen of the terminal bronchiole communicating with an alveolar cyst is scarred by old disease such as tuberculosis, or narrowed by fresh infection, as seems to happen in staphylococcal pneumonia, air may be admitted during inspiration when the bronchi dilate, but is unable to escape when they contract on expiration. The cyst consequently grows in size with every breath or at least with every deep inspiratory gasp such as that preceding coughing (see Fig. 1). As its size increases it compresses neighbouring tissues more and more, including the bronchi which supply it, and whose entry into the cyst becomes therefore more and more oblique. This in itself operates as a kind of ball valve, and one that constitutes a vicious circle, becoming worse as the cyst gets bigger. The rest of the lung is squeezed away by the ballooning cyst, the breathless patient gasps harder, the cyst grows bigger still. It may fill the thorax and displace the mediastinum, calling for emergency relief. Most cysts, however, eventually reach a state of equilibrium when the positive pressure within them is sufficient to force a leak of air back into a bronchus. The size then remains fairly stable increasing only as neighbouring smaller cyst-spaces break down and fuse with it. If free communication exists between the cyst and the bronchus the patient may still be disproportionately breathless because so much of his tidal air is simply blowing in and out of the cyst instead of becoming oxygenated elsewhere (X ray 41)

Alveolar cysts are divided into *blebs* and *bullae* blebs being defined as subpleural collections of air caused by the rupture of alveoli directly beneath the visceral pleura or by interstitial air leaks and bullae resulting from the coalescence of one alveolus with another. The distinction is largely an artificial one, but whereas blebs are often seen clustering round apical adhesions or old tuberculous scarring, and may be associated with purely local stresses bullae suggest more generalized tissue damage. The surgical importance of both is (1) their tendency to increase in size, and so cause breathlessness, and (2) their tendency to burst, and so result in spontaneous pneumothorax.

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is particularly mobile) into the opposite chest, and resulting in laboured breathing and cyanosis which can sometimes be relieved by the insertion of a needle into the cyst and the application of sufficient suction upon it to re-expand the lung. A congenital origin used to be postulated for these vesicles, but evidence for this is lacking, although an inborn pulmonary defect may account for some of them. It is probable that the great majority are in fact the remarkable thin-walled cyst-abscesses which accompany *staphylococcal infections* of the lung, and are particularly common and dramatic in early childhood. Terminal bronchioles become partially obstructed so that a group of breaking-down alveoli beyond are subjected to sudden positive tensions causing them to expand into large pseudocysts, which may contain a fluid level of staphylococcal pus, but are often quite empty. Many rupture into the pleural cavity at an early stage, contaminating it and resulting in pyo-pneumothorax, but frequently the positive pressure element causes most trouble, and not the infection, for although the cyst has ruptured, its bronchial check-valve mechanism continues to function but is now applied through the cyst to the pleural cavity. Very often the initial pneumonitis passes quite unnoticed until the mechanical effects of a ballooning cyst direct attention to the chest. It is very rarely that any surgical intervention is called for beyond the insertion of a needle and the application of suction. Re-expansion of the lung and closure of the leak is the therapeutic object whether the pleural cavity is contaminated or not. Penicillin is of course given whenever a staphylococcal infection is in question.

Young adults between the ages of 20 and 25 are particularly subject to *spontaneous pneumothorax* as a result of rupturing blebs or bullae. A high proportion of such pneumothoraces become *chronic*, the lung failing to re-expand within three months; whereas if the cause persists and leaks again, they are *recurrent*. In conditions affecting both lungs they are likely to occur first upon one side and then upon the other. If the leak is valvular a *positive pressure pneumothorax* calling for emergency relief develops (X-ray 14). Blebs and bullae in this age group, men being affected eight times more often than women, are due to the following two main causes, of which the first is the commoner.

1. *Healed apical tuberculous scarring* such as can be found in most peoples' lungs, usually, but by no means always, confined to one side. This cicatrization distorts the terminal bronchioles, giving rise to groups of little blebs, and at thoracoscopy these are seen clustering at the base of apical adhesions. Even when spontaneous pneumo-

thorax complicates active tuberculosis, as it not infrequently does, such blebs are often the cause. Usually spontaneous pneumothorax due to old apical scarring is unilateral, even when recurrent. The chances of such blebs rupturing first on one side and then upon the other are clearly not great.

2. Bilateral pneumothoraces, especially if they are recurrent, are almost always due to *generalized emphysema*. Pathologically this is indistinguishable from the chronic degenerative emphysema so commonly seen in the aged in which also there is general loss of pulmonary elastic tissue but clinically it is a clear-cut entity affecting otherwise healthy young adults, and may be due to a congenital deficiency of the lung's elastic fibres. Some support for this view is given by the fact that if an affected lung is viewed early in the disease either at thoracoscopy or thoracotomy small blebs are visible scattered over its surface, quite unrelated to scars, and fed by minute air bubbles seething up between the alveolar lobules in a way that strongly suggests an interstitial defect. At this stage no bullae are to be seen at all. Later on bullae do appear, usually at the apices and radiologically one such cyst may be so prominent that it is thought to be solitary. Thoracotomy demonstrates this almost never to be the case and although one large cyst may compress the rest of a lung and be responsible for most of the symptoms, it is very rarely indeed that the remaining lung is found to be quite healthy or the large bulla to be unaccompanied by others less conspicuous.

Patients with this condition tend to be tall and thin and to have flattened chests. Sometimes they give a history of asthma or recurrent chest infections, or may acknowledge themselves to be short of breath but quite frequently the occurrence of a pneumothorax is their first indication that anything is amiss.

'Compensatory' emphysema, secondary to atelectasis, or to the removal of an adjacent lobe or obstructive emphysema due to bronchial stenosis, may also give rise to blebs and bullae, and so to pneumothorax.

Spontaneous pneumothorax

Spontaneous pneumothorax is usually sudden in onset and accompanied by stabbing pain which may be mistaken for pleurisy, lumbago or even a perforated peptic ulcer. The patient is often short of breath, but very seldom cyanosed unless a tension element is present. The accident sometimes follows exertion, although this can be of the most trivial sort, such as getting out of bed. Occasionally the onset is insidious, and the pneumothorax discovered only on investigation.

and in any case the symptoms depend to a large extent on the degree of passive collapse of the underlying lung, which may merely be separated from the chest wall by a thin film of air, or flattened against the mediastinum. For this reason physical signs of pneumothorax (diminished movement, increased resonance, decreased breath sounds, and mediastinal displacement to the opposite side) are not always reliable, and the confirmation of a radiograph is necessary (X-ray 15).

If the rupture of a bleb has been associated with bleeding, either from the lung or from an adhesion torn by the sudden collapse, a fluid level is present and the condition becomes one of *spontaneous haemopneumothorax* (X-ray 13). Indeed the thorax may be filled with blood, displacing the mediastinum and exsanguinating the patient so that an immediate thoracotomy is necessary to remove the blood, stop the bleeding, and re-expand the lung.

The other circumstance under which a fluid level is visible is when the escape of air is due to the rupture of a tuberculous cavity, usually after the induction of an ill-judged artificial pneumothorax, or incomplete adhesion section, in which case a tuberculous empyema will swiftly follow unless emergency pulmonary resection is undertaken, and the condition is really one of *pyopneumothorax*.

Treatment

In at least 80 per cent of cases no fluid is present in the pleural cavity, and the cause of the air escape is one or other of the two common sources of blebs or bullae already described. If the attack is the first one, treatment consists simply of inserting an A.P. induction needle of the Kuss type (that is, one without a sharp point) and removing the air in the pleural cavity by means of an artificial pneumothorax apparatus. Should it be found that a negative pressure is not maintained, it is obvious the hole in the lung must still be leaking, and an indwelling needle of the Foster-Carter type is substituted, and connected with an underwater-sealed drain and continuous suction. It is then possible to see, on turning off the suction, when the leak has healed, for fluid from the drainage bottle will rise up the closed tube, indicating a negative pressure in the chest; whereas if a leak persists, air continues to bubble out of the tube into the bottle. An indwelling needle is not satisfactory for more than 48 hours, so after that period a soft rubber catheter is inserted instead, with a suitable trocar and cannula, through the second or third intercostal space anteriorly, under local anaesthesia. This can be used for much

longer, and suction is maintained on it for 36 hours or so after X rays show the lung to have fully re-expanded.

In the great majority of cases the simple removal of air is all that is required but should this not be the case, either because of continued leaking, or because the pneumothorax is a chronic one, and the lung imprisoned by thickened pleura, thoracotomy is indicated. The longer such a lung remains collapsed the less likely is it to re-expand. Furthermore, in some 20 per cent. of pneumothoraces recurrence is likely within a year or less, and such chests should always be explored. Quite frequently a history is given of repeated attacks, first upon one side then upon the other so that the patient lives in constant dread and is afraid to work. One such patient of mine was a steel worker who had had three attacks upon one side, two upon the other and was no longer willing to ascend scaffolding. Another was a young medical orderly who had had seven attacks on the right, five upon the left, and had undergone a variety of treatment (including chemical pleurodesis) on both, followed swiftly by recurrences. These and many similar patients are permanently cured at thoracotomy and, most important of all, can be given unequivocal guarantees that their troubles from spontaneous pneumothorax are at an end. The treatment of such pneumothoraces by inducing a chemical pleurisy with insufflated iodized talc or 10 per cent. silver nitrate, and subsequently withdrawing air in the hope of making the inflamed visceral and parietal pleural layers adhere has nothing to recommend it. It is highly unreliable, recurrences after it are common, and it is much more disabling and painful than thoracotomy besides leaving the true nature of the lesion still in doubt. Thoracoscopy is likewise an inadequate substitute for direct examination and cure.

An intercostal thoracotomy should be performed and a search of the surface of the lung made for the cause. In unilateral cases this is likely to be a small bleb related to an apical adhesion or to old scarring, and the leaking hole in it may be readily visible. The base of the bleb or bunch of blebs, is clamped, cut, and the raw area oversewn. If the rest of the lung is perfectly healthy it is re-expanded and the chest closed.

In others and usually in bilateral cases, blebs or bullae are found scattered over the surface of the lung, quite unrelated to scars but evidently the result of generalized changes in the lung itself. A froth of tiny air bubbles can often be seen working its way to the surface between the lobules. In some a ruptured bleb is obvious, in others it must be sought for like a leak in a bicycle tyre and when found under run with a mattress suture. As further leakages are inevitable

if the lung is left free, the whole layer of the parietal pleura is stripped off the chest wall and the mediastinum, and all bleeding points coagulated. Two underwater-sealed drains are inserted, one at the apex to remove air, one at the base for effusion, and both connected to continuous suction for at least 48 hours. The lung is fully expanded by the anaesthetist and the chest closed. The lung thus becomes firmly adherent to the chest wall, and the patient can be given assurance that his condition will never recur.

PNEUMATOCOELES

Bullae in young adults may give rise to symptoms not by bursting but by bulk. They grow, partly by coalescence with neighbouring bullae, but mainly by the increasing tension within caused by a check-valve mechanism, so that one vastly outgrows the rest and compresses the healthier parts of the lung. Radiologically it is difficult to distinguish between a large cyst of this type and a localized pneumothorax, for there is much increased translucency and absence of lung markings in both, but fine trabeculae are generally distinguishable, especially at the base of a cyst, and the curvature of the cyst edge can be seen as it comes in contact with the chest wall (X-ray 39)

Giant bullae of this kind should be excised locally if possible, that is, if sufficient healthy lung is left around them. They have, of course, no lining membrane, and no wall except the compressed and atelectatic surrounding parenchyma. This condensation can be dissected out and the cyst almost enucleated, but at its base are always a number of bronchial openings which must be carefully closed (X-ray 40)

In other cases it is found that the pneumatocoele completely replaces the tissue of a lobe, or that the rest of the lobe contains other bullae almost as big. Lobectomy is then the best treatment, not only by getting rid of the cyst's dead space and so allowing the rest of the lung to re-expand, but also by re-routing the blood supply to more functional areas and thereby increasing its oxygenation.

Such operations are frequently of great benefit, doing much to relieve dyspnoea and enabling patients to work and lead active lives; but it must not be forgotten that the lung left behind is defective, and the eventual prognosis from this point of view is bad.

The following is a fascinating example of the condition and illustrates several features of the disease. A tall thin police sergeant of 37 was sent up after routine radiography had revealed a 'giant cyst' at the apex of his right lung. The X-rays showed not only a giant

bullae at the right apex but a suspicious increase of translucency at the left otherwise his lung fields were normal. He was a non smoker was on full active duty, and had never been short of breath. In view of this he was reluctant to have an operation, and I agreed to observe him for the time being. No change occurred, either in the size of the cyst or in his condition for two years but at the end of this time he admitted to some breathlessness and agreed to have his cyst excised. His lung fields were then still clear, but on admission X rays showed an opacity in the midst of the hypertranslucent left upper lobe, and this was at first thought to be an inflammatory lesion of the type common in such lungs and was treated with antibiotics (X ray 37) It did not resolve, and bronchoscopy proved it to be an early squamous-celled carcinoma arising in the left upper lobe bronchus. Here indeed was a dilemma lobectomy on the left side was out of the question in view of the pneumatocoele upon the right, and in any case it was doubtful if the growth could be extirpated by lobectomy alone. Bilateral surgery offered the only solution. Deep X ray therapy was begun in order to diminish the neoplasm as much as possible, though squamous growths are not usually very responsive. At right thoracotomy it was found the upper lobe consisted only of the giant cyst and several others and it was therefore removed. The lower and middle lobes showed marked emphysematous changes, and the leakage from their raw surfaces was so severe that a positive pressure pneumothorax developed postoperatively upon this side in spite of the use of continuous suction from a Roberts pump because the lung was leaking faster than the pump sucked! (X ray 14) It was not until three such pumps were working together that the remaining lobes re-expanded satisfactorily and filled the thorax. After a fortnight's rest, a left thoracotomy was performed, and the growth which had shrunk considerably in the interim, was successfully removed by left upper lobectomy. Some weeks later the left lower lobe began to show signs of increasing emphysema due to stretching, and a left apical lateral thoracoplasty was done to preserve it. The patient, whose unflinching courage alone made this gruelling programme possible remains well, active and in good spirits (X ray 38)

In elderly men chronic degenerative emphysema in some degree or other is almost universal and is often associated with chronic bronchitis or with a raised blood pressure. It is thus one of the commonest causes of haemoptysis, especially when hyperpiesis is also present, for bleeding occurs when alveolar walls break down. Although the degree of emphysema may be severe, and bullae evident in the lung, spontaneous pneumothorax is surprisingly rare. This is partly

because with the passage of time the layers of the pleura have become adherent owing to repeated mild inflammatory attacks, but even more because the connections of the cyst with the bronchi are wide and numerous, and so less likely to become valvular. Nevertheless pneumothoraces sometimes occur, and when they do so are more likely to persist and become chronic. The pleura thickens over the collapsed lung which then cannot re-expand even if the leak heals. At thoracotomy this thickened crust can be peeled off, and expansion restored. Occasionally it is justifiable to excise a large bulla that is interfering with pulmonary function, but the probable state of the rest of the lung must always be borne in mind (X-rays 39, 40).

HYDATID DISEASE OF THE LUNG

The parasitic worm *Taenia echinococcus* dwells in the intestine of the dog and is about 0.5 cm long, its intermediate hosts are sheep, pigs or men, in whom it goes through its larval and encysted forms. Dogs become infested in sheep-farming countries by eating sheep's offal, and man either by direct contact with dogs, or from eating lettuce or other produce contaminated by their faeces. Hydatid infestation is rare in England, but common in Wales, in the Argentine, some parts of the Middle East, Iceland, Australia and New Zealand.

Man ingests the ova discharged from the worm's terminal segment, and after gastric juices have digested their capsules, the embryo, or *hexacanth* (as it is called from its corona of six hooks) transverses the stomach wall, enters the portal vein, and reaches the liver where about 75 per cent of cysts are found. Should it escape this filter it finds itself in the inferior vena cava, and so passes via the right heart into the lung where it lodges and enters a larvated stage. Up to 25 per cent of cysts may therefore appear in the lung. Infestation of more than one organ occurs in less than 2 per cent of cases, and some 2 per cent involve the heart, sometimes causing paroxysmal tachycardia.

The embryo now vacuolates and forms a small vesicle composed of two walls: an inner, nucleated germinal layer, and an outer, non-nucleated layer. This vesicle expands into a cyst, at first small, but becoming progressively larger if it is undisturbed. From the germinal layer groups of cells bud off to vacuolate in turn and form 'brood capsules' which give rise to *scolices*, the heads and source of new worms. Most of these become detached, and lie at the base of the cyst bathed in hydatid fluid. Outside the cyst the lung reacts to it by condensing a *pericyst*, or adventitious layer, of fibrous tissue.

round it, which can be easily separated from it as is the skin from a banana.

Up to this stage the hydatid cyst has produced no symptoms though it may be revealed on routine radiography as a spherical opacity with a clear-cut edge (X rays 43-44). It is usually solitary but sometimes more than one is present. The Casoni test a skin reaction to the intradermal injection of hydatid fluid, is diagnostic when positive but is not always reliable when negative and an eosinophilia of 4 per cent. or more is strong supporting evidence.

The fate of the cyst and the symptoms it causes now depend largely upon the complications which overtake it. As it increases in size it sometimes causes pain or dyspnoea, and occasionally anaphylaxis, with severe bronchospasm, breathlessness and tachycardia, although this is more usually seen after rupture or surgery.

The continued expansion of the cyst tends gradually to cause pressure erosion of a neighbouring bronchus, so that finally a fistula is established and air escapes into the adventitious pericyst, stripping the true hydatid from it. If the cyst is small it may now be expectorated whole, and the patient thus spontaneously cured or it may rupture, and the diagnosis is established when the patient spits up hooklets, scolices or fragments of laminated membrane. By rupture into a bronchus or into the pleural cavity scolices are spread and multiple cysts develop. pleural rupture resulting also in a hydro-pneumothorax. Pulmonary vessels are sometimes eroded in the same way. Multiple cysts of the lung occur in about 6 per cent. of cases and are commoner in children. Rupture itself carries a high mortality.

Once a fistula is established ultimate infection of the cyst is inevitable, although this does not necessarily kill the hydatid, which may continue to bud off daughter cysts. Even when the main cyst dies, daughters can survive among the pus. They are more commonly associated, however with liver infestation and should they appear in the sputum hepatic involvement is to be feared, for infected liver cysts can perforate the diaphragm and rupture into the lung or the pleural cavity. As well as these consequences, infection brings with it fever, purulent sputum, haemoptysis and general evidence of pulmonary supuration.

It is important to bear in mind that hydatid cysts seldom die except as a result of rupture or infection and not always then. Even calcification of the cyst wall does not necessarily imply the death of the parasite.

Treatment

The treatment of hydatid cysts of the lung is surgical, and has for its object the complete removal of the parasite with maximum conservation of pulmonary tissue. Expectant treatment, attempts at transpleural aspiration, the induction of an artificial pneumothorax, or even bronchoscopy, are all contra-indicated lest they result in rupture and dissemination.

After accurate radiographic localization of the cyst, the chest is opened where direct access to it can best be obtained. The pleural space is carefully packed off, some surgeons using black towels to detect contamination more easily, or tying a macintosh funnel round the affected lobe. A fine-gauge needle attached to a two-way syringe is passed directly into the cyst and some of the fluid slowly aspirated. A small incision is then made down to the pericyst until the friable, glistening, bluish-white laminated membrane of the hydatid itself is visible and can be removed complete, meticulous care being taken not to rupture it or to spill any of its contents. The cyst has in any case a natural tendency to herniate out of the lung once the pericyst is incised, and this can be increased by the anaesthetist softly raising the pressure in his circuit.

The interior of the pericyst is inspected, bleeding points are secured, and any fistula present closed. The cavity is soon obliterated as the lung re-expands. Multiple cysts are dealt with in the same way.

If a cyst has already ruptured and is infected, or should it be associated with bronchiectasis or with serious bleeding, lobectomy may be necessary; but every effort should be made to conserve as much healthy lung as possible, and if local removal of the cyst can be achieved it is to be preferred, both pericyst and pleura being subsequently drained.

INNOCENT TUMOURS OF THE LUNG

IN comparison with bronchial carcinomas innocent tumours of the lung are rare, and in a great many cases are removed only because their identity is in doubt but by other standards the two chief varieties are by no means uncommon adenomas of the bronchus accounting for 4 per cent. of all lung neoplasms, and cartilaginous hamartomas occurring about once in every 300 autopsies Further more, though most hamartomas are symptomless, adenomas invariably produce serious symptoms and lung destruction in time, apart from their being subject to some risk of malignant change, and their early diagnosis and prompt excision is thus of prime importance

BRONCHIAL ADENOMAS

Adenomas are eight to ten times commoner in women than in men, thus exactly reversing the sex incidence of bronchial carcinoma. They grow from the mucus-secreting glands in the deeper layers of the bronchial mucosa, almost always that lining the walls either of a stem bronchus or of one of the main lobar bronchi, with the important practical consequences that as they increase in size they cause lobar collapse and are bronchoscopically accessible They also extend into the surrounding lung, and this portion is generally much bigger than that visible in the bronchial lumen.

Histologically they consist of cuboidal cells often having a regular acinar arrangement with evidence of mucus secretion, but sometimes constituting apparently disorderly cellular masses, the cell clumps being held together by highly vascular connective tissue Superficially these are easily mistaken for anaplastic carcinomas, but mitotic figures are rare, and the part of the tumour presenting in the bronchus is covered by a layer of bronchial mucosa. Much wearisome debate has raged as to whether adenomas are wholly innocent or eventually of low-grade malignancy It is true that a few rare examples of metastasis from them are on record but the clinical course of the overwhelming majority is quite innocent, and there are many cases in which the tumour has been present in the lung for forty years or more without dissemination As they all used to be subjected to repeated bronchoscopic fulguration in order to clear the bronchial lumen, it is not surprising that malignant changes occasionally

supervened; and it is perhaps best to think of them as resembling mixed parotid tumours in this respect. What is certain, however, and very important in practice, is that surgical removal of adenomas should be as conservative as possible and it is a tragedy if a whole lung be removed because of a mistaken interpretation of the histology when perhaps a bronchotomy or at most lobectomy might have sufficed.

When a young woman with a sound heart coughs up bright blood the first disease to be thought of is tuberculosis, the second bronchiectasis, and the third a bronchial adenoma. The patient is generally between 20 and 40, and gives a history of repeated haemoptysis spread over several, or many, years. Bleeding may be profuse, and because of the age at which it occurs may lead to a long and fruitless search for tubercle bacilli or even to the patient being immured in a sanatorium.

A patient of mine had an haemoptysis when she was 22 and was referred at once to a chest clinic where an X-ray showed her to have a collapsed right lower lobe. Her sputum, which was purulent, was searched for tubercle bacilli; and though none were found the condition was diagnosed as pulmonary tuberculosis and she was told to attend the clinic regularly. After a time she developed a right empyema and was admitted to a general hospital where it was drained (X-ray 45). After the drainage tube was removed from her chest a sinus persisted from which pus continued to trickle; and so she drifted for nearly five years from chest clinic to chest clinic, and hospital to hospital, having occasional haemoptysis, her X-ray substantially unchanged, and pus oozing constantly from her side. At no stage did anyone bronchoscope her, and eventually she produced tubercle bacilli in her sputum, for she had meanwhile developed tuberculosis at the apex of her right lung. She was given streptomycin, and finally referred for the treatment of an 'unhealed tuberculous empyema' (X-ray 46). Bronchoscopy revealed an adenoma completely obstructing her right main bronchus just below its upper lobe branch, and pneumonectomy rid her of her growth, her empyema and her tuberculosis.

As adenomas grow in main bronchi, bronchial obstruction with distal collapse of the lung is the next most likely source of symptoms. Once obstruction has occurred infection is usual, giving rise first to purulent sputum and bouts of fever and malaise, which may be interpreted as 'pneumonia', 'influenza' or 'bronchiectasis'. Later, as the blockage becomes more complete, expectoration ceases, but the infection may instead cause an empyema. If there is no infection, the

dammed up mucus distends the bronchus into a mucous cyst, and as the growth can bleed distally as well as proximally there may be a large haematoma of the lung distal to it.

A boy aged 16 had an attack of left sided pleurisy with fever, and was admitted to a general hospital where an X ray showed opacity of his whole left lung. He was given antibiotics and the shadowing cleared. This episode was repeated again and again during succeeding years and altogether he was in hospital seven times in three or four years. As he began to spit up yellow phlegm towards the end of this period bronchograms were done which showed some bronchiectasis in his left lung for which he was referred for treatment. They also showed a constant and peculiar filling defect in the left main bronchus, and subsequent bronchoscopy proved this to be due to a bronchial adenoma almost filling the lumen and which had been responsible for the episodic collapses (X ray 47)

Bronchoscopy is essential to diagnosis and as most of the growths are within bronchoscopic range a biopsy can nearly always be obtained for histological confirmation. The adenoma appears as a lobulated raspberry like tumour and quite sharp bleeding from it is apt to follow the biopsy. If this occurs the head of the operating table must be lowered so that the blood will run down the bronchoscope and not obstruct the patient's airway or be aspirated into his lung. At the same time suction through a wide-bored tube is applied and as soon as a view can be obtained of the growth a swab soaked in adrenaline is passed and held against it until the bleeding stops.

Once an adenoma has been identified, surgery is indicated, and must be as conservative as possible although no place exists for the bad old practice of fulguration, or for the insertion of radon needles, to which adenomas are quite insensitive. In exceptionally early cases it may be possible merely to open the posterior wall of the bronchus and shell out the tumour with the cuff of mucosa round its base but as most patients unfortunately already have a long history and lung damage secondary to obstruction has occurred, a lobectomy or even a pneumonectomy is usually necessary. The latter should never be performed when the former is practicable and the results of conservative resection are excellent.

HAMARTOMAS

This title which implies a developmental error, is loosely used to include two quite different types of innocent tumour brought into considerable prominence by mass radiography one in which cartilage greatly predominates, and one that is essentially a vascular

abnormality Both are usually symptomless, and their importance largely lies in the difficulty in distinguishing them from small round carcinomas; but when the vascular ones include an arterio-venous shunt of blood their effect is likely to be profound.

Cartilaginous hamartomas

Cartilaginous hamartomas are comparatively common (28 being found in a series of 8,800 post-mortems at the Mountefiore Hospital), and occur three times more frequently in men than in women, nearly always peripherally in one or other of the lower lobes. They are a mixture of mesodermal and epithelial elements, being mostly composed of cartilage but containing also fibrous connective tissue (often myxomatous), cuboidal or columnar epithelium, distorted glands, fat and smooth muscle. A definite capsule is not present although they are sharply circumscribed and can readily be shelled out of the adjacent lung. In the past they were met with only at autopsy and called 'chondromas of lung', but pure chondromas probably do not exist. When calcification occurs in the cartilage it is eccentric and lobular, and radiologically very characteristic (X-ray 48); but in its absence, that is in the great majority of cases, it is quite impossible to be sure they are not early peripheral carcinomas, and their removal becomes imperative. Even at thoracotomy the distinction may not be easy. As I was enucleating one its edge began to break and appeared very like a neoplasm. A lobectomy was performed, and the pathological report was that although the tumour was a hamartoma its edge appeared to be growing invasively; and Simon and Ballon also reported a case in 1947 with some suggestion of malignancy. But I do not want to give the impression that this often happens. As a rule the diagnosis is readily made, and the tumour feels much harder than does so small a carcinoma, is smooth, lobulated, and curiously mobile within the lung parenchyma, which should be incised directly down upon it. Its white glistening surface then appears and the whole hamartoma can be shelled out, after which the bleeding points in the lung, controlled by digital pressure, are individually tied, or coagulated, and the raw surfaces approximated. If any doubt about the nature of the tumour exists a frozen section will settle the matter.

Vascular hamartomas

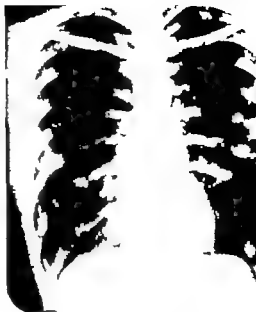
When these are symptomless no differential diagnosis can be made without thoracotomy, but sometimes a history of familial telangiectasia or the presence of telangiectases on the lip or mucous mem-



41

An emphysematous bulla with calcification in its walls. It occupies a large part of the pleural space and the rest of the lung.

A staphylococcal cyst-abscess at the base of the left lung in a boy who had just had a cerebral abscess. It is exactly like an emphysematous bulla, except that it transiently contained very little pus, and it rapidly as it came. Its behaviour was decided by the air pressure within it.



42



43

A hydatid cyst in the lower lobe of the left lung.

Lateral view of a hydatid cyst in the lower lobe of the left lung. Before admission this was mistaken for carcinoma. (Compare with X-ray 99)

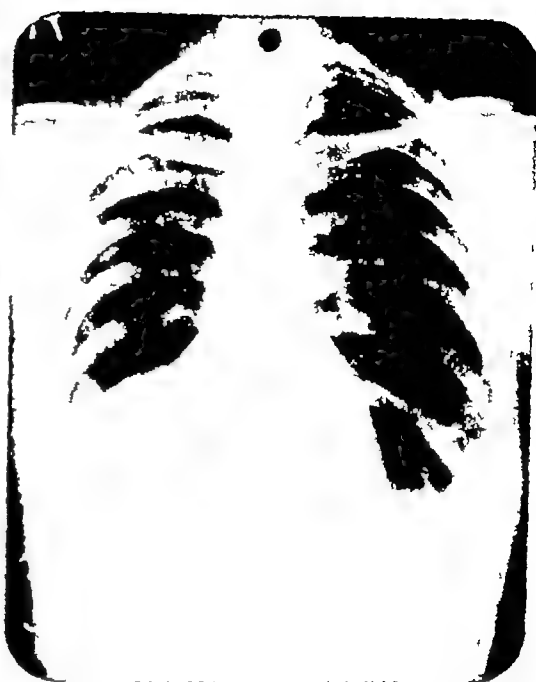


44



45

X-ray 45 A right empyema in a young woman who suffered from recurrent haemoptysis. Tuberculosis was diagnosed. After drainage the right lower lobe was seen to be collapsed (See X-ray 46).



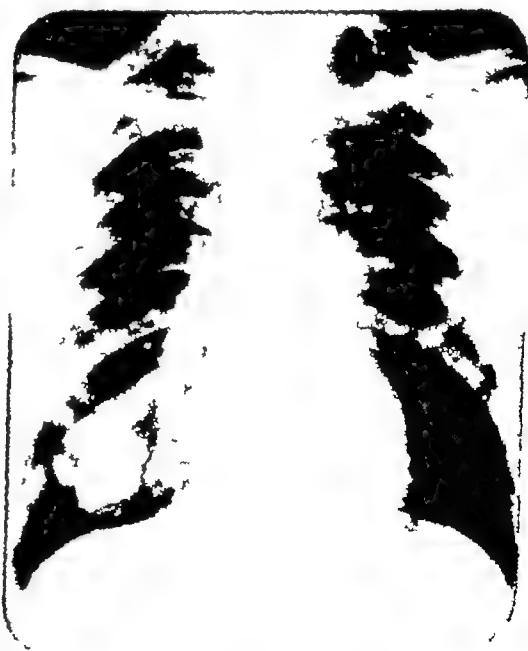
46

X-ray 46 The empyema seen in X-ray 45 was secondary to the collapse of the right lower lobe seen here. This in turn was caused by a bronchial adenoma. Secondary tuberculosis is now also visible at the right apex; none was present at the time of the empyema. Pneumonectomy was performed.



47

X-ray 47 In this otherwise poor bronchogram of the left lung, as well as bronchiectasis of the lower lobe, the dye has outlined a substantial filling defect arising from the roof of the left main bronchus. It is a bronchial adenoma, the bronchiectasis being secondary to the intermittent obstruction caused by the growth.



48

X-ray 48 A calcified chondromatous hamartoma in the lower lobe of the right lung.

branes of the mouth are suggestive and are especially likely to be found when there are multiple lesions in the lung. One vascular hamartoma I removed was simply a blind aneurysmal dilatation of an abnormal pulmonary vessel, appearing as it was shelled out like a blue pulsatile plum.

On the other hand a single lesion more like a cavernous haemangioma, may have within it an arterio-venous fistula connecting a pulmonary artery and vein and the tortuous distention of the adjoining vessels gives rise to an X-ray shadow which on screening or kymography is seen to be pulsatile. The short-circuiting of unoxygenated blood from the pulmonary artery directly into a pulmonary vein, and so to the systemic circulation, has serious consequences for as much as half the blood volume has been known to be shunted in this fashion. The results are cyanosis, of the central type, breathlessness, polycythaemia and clubbing of the fingers and toes—a combination of features which otherwise strongly suggests a congenital heart defect. If the chest is auscultated over the lesion a systolic murmur can sometimes be heard, which, in contrast to all cardiac systolic murmurs, gets louder on inspiration as blood is sucked into the hamartoma by the concertina like action of the lung. This syndrome is an urgent indication for excision of the lesion, if necessary by lobectomy.

The only other innocent tumours that are found in the lung are all exceedingly rare but include *intrapulmonary neurofibromas* growing from nerve branches associated with the bronchi.

BRONCHIAL CARCINOMA

THE explosive increase in cancer of the lung during the past decade or so has made it one of the major social problems of our time (Fig. 26) It has reached almost epidemic proportions in the male

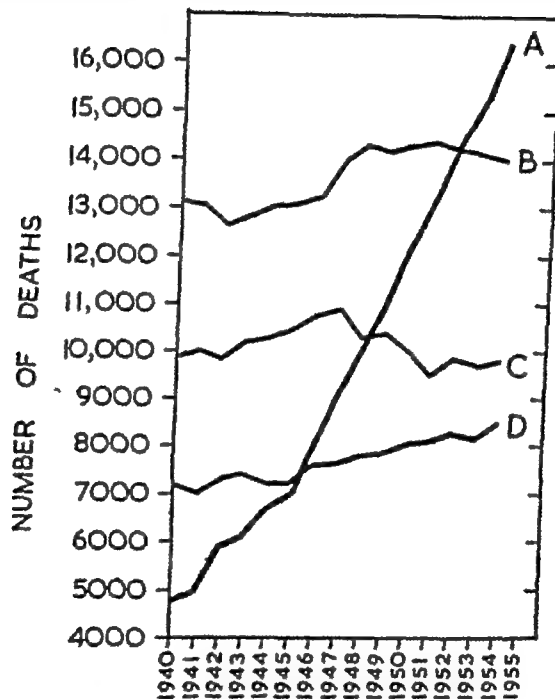


FIG 26

Graph (after Horace Joules) showing the rise in deaths from cancer of the lung in England and Wales between 1940 and 1955.

Deaths from cancer of lung (A), stomach (B), intestine (C), and heart disease (D)

population over 40, and has supplanted pulmonary tuberculosis as the commonest serious lung disease. The extent of this increase, its significance, and its cause, have become in recent years the subject of much publicity and controversy. What are the facts?

Incidence

Cancer of the lung is now the commonest form of cancer, and overwhelmingly the commonest variety in the male. More than 80

per cent. of the victims are at present men, the disease being responsible for 40 per cent. of all male deaths from cancer and for more than 7 per cent. of male deaths from all causes but the proportion of women affected shows some signs of rising.

In the 25 years between 1922 and 1947 the number of deaths in England and Wales due to it rose fifteenfold, from 612 to 9,287, this increase being out of all proportion to the increase in population. In New York State which has particularly well kept records, the rate has leapt up 50 per cent. in men and 55 per cent. in women and has been sufficient to raise the overall male cancer rate by 21 per cent.

It may be objected that much of this increase is apparent rather than real, as it is only in the past 20 years that proper diagnosis of chest disease has existed and the generation of family doctors over the age of 40 were taught extremely little about lung cancer in their medical schools.

In the last decade however doctors have been alert to the condition, and diagnostic methods have not notably improved. Yet in 1944 in England and Wales there were 6,584 deaths, 5,347 in men and 1,237 in women, and in 1955 17,272, of which 14,821 were male and 2,451 female. The London County Council reported 863 deaths in 1944 1,785 in 1954. These increases, which are closely paralleled in other civilized countries, may therefore be accepted as real. Further evidence of this is that the male death rate in 1955 was 13 times that in 1930 while the female rate increased only 5 times in the same period, a difference which cannot be accounted for by better diagnostic methods. Furthermore the relentless rise continues unabated.

The World Health Organization reports for 1952, the last year for which full figures are available the following death rates per 100,000 from lung cancer in men, with the percentage rise in four years (that is, from 1949)

England and Wales	61.4	(up 31%)
Scotland	56.3	(up 36%)
Ireland	22.2	(up 47%)
Italy	61.4	(up 45%)
Netherlands	30.3	(up 24%)
France	28.2	(up 30%)
Switzerland	33.5	(up 28%)
Finland	38.7	(up 30%)
Denmark	24.8	(up 49%)
Australia	20.8	(up 25%)
New Zealand	31.5	(up 46%)
Japan	4.9	(up 68%)
United States	26.1	(up 21%)

It has been suggested that an ageing population inevitably increases the rate of cancer incidence: but the increase in cancer of the lung is wholly out of proportion to this element. Although bronchial carcinomas occur at any age between adolescence and senility (a few patients being seen in their twenties, and many in their thirties and seventies), the most affected are those between 45 and 65, not those in the later decades; and, most conclusive of all, no other form of cancer has increased comparably, carcinoma of the breast remaining

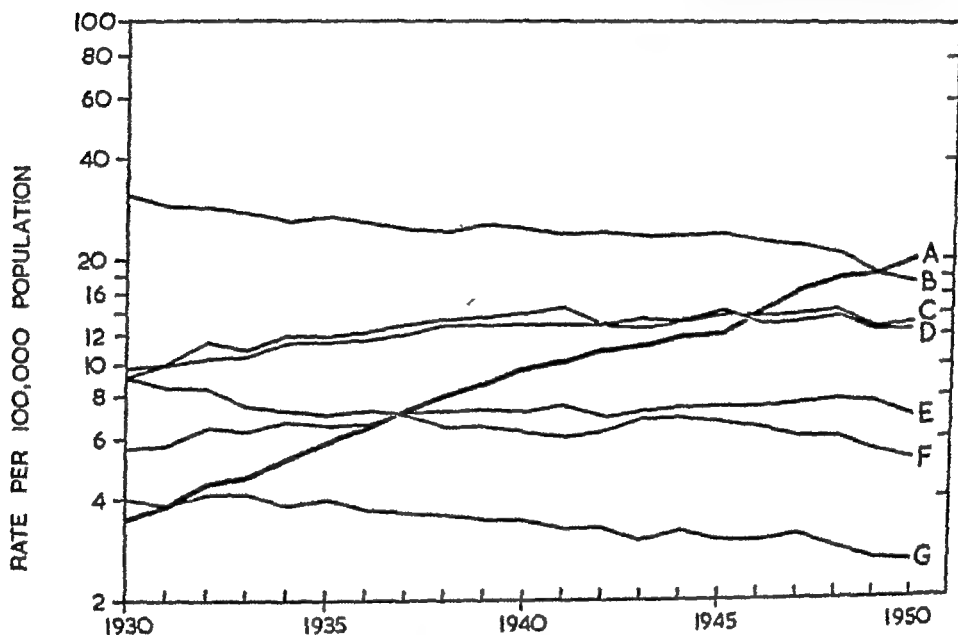


FIG. 27

Graph (after Evarts Graham) showing the rise in deaths from cancer of the lung between 1930 and 1950 in the U.S.A. compared with deaths from other forms of cancer.

Deaths from cancer of lung (A), stomach (B), prostate (C), intestines (D), rectum (E), liver (F), and skin (G), among white males.

stationary, and the rates for cancer of the stomach, rectum and uterus having declined. The situation in the United States is very similar (Fig. 27).

The incidence of carcinoma of the lung is, at present, notably higher in large cities than in the country, the death rate in London being twice that in rural districts; and this difference has also been reflected in the United States and elsewhere. Eastcott has recently (1956) demonstrated that immigrants to New Zealand from the United Kingdom have a 30 per cent. greater chance of dying from lung cancer than have the natives of that country, and the risk is

75 per cent greater if the migrant was over 30 on entry thus strongly suggesting that the English and Scots were conditioned by their former environment, and that the effect was related to the length of their exposure to it.

Aetiology

In 1912 Adler suggested that cancer of the lung might be caused by tobacco smoke. The evidence that it is a factor of prime importance is now overwhelming. The rise of bronchial carcinoma everywhere in the world has closely followed the consumption of cigarettes, and, so far as can be seen at present, no other factor but the carcinogens they contain require some 20 years to produce their effect. A great increase in cigarette smoking occurred during and just after the First World War and between 1910 and 1925 consumption rose by 100 per cent. It is the men who began smoking in 1914-18 who developed their pulmonary neoplasms about the time of the Second World War. The higher a country's cigarette consumption 20 years ago the higher its lung cancer mortality today. In the decade 1891-1900 the annual consumption of cigarette tobacco per adult man was 0.4 lb. During 1911-20 it went up to 3.8 lb. the First World War having intervened. In 1941-50 it reached 8.3 lb. The difference in female consumption is striking to all intents and purposes they smoked nothing at all before the decade 1921-30 and then only 0.2 lb per year per adult woman. Even during the second war decade 1941-50 it reached only to 2.4 lb. that is less than men smoked in 1911-20. They are in short, 30 years behind men in cigarette consumption and this may well account for the disparity of 8:1 in the sex incidence of bronchial carcinoma.

This 20-year lag must be considered when it comes to accounting for the difference between the cancer rates of town and country. Although chain smoking is commoner in urban and industrial communities and the smaller a man's home town the less he smokes this is today not enough to explain the facts. The very different smoking habits in rural districts of 20 or 30 years ago might very well do so as they may also help to explain the greater susceptibility of British immigrants to New Zealand.

The difference between town and country, industrial homeland and pastoral colony must nevertheless raise the question of whether air pollution by industrial smoke, tarred roads, and petrol or diesel exhaust fumes, play any part in the aetiology. The fact that both sexes are on the whole, equally exposed to the prevailing atmosphere but do not share the disease to anything like the same extent, cannot

be explained away. There is in fact some evidence that atmospheric pollution has actually decreased since the end of the nineteenth century. Furthermore no evidence of any kind has been produced to inculpate these fumes, beyond the fact that the carcinogens present in tobacco smoke are also to be found, albeit in much smaller concentrations, in them. Men constantly exposed to heavily polluted air and exhaust fumes, as are some factory workers, garage hands, policemen and transport drivers, do not have a high cancer rate, nor is the comparative immunity of the urban-dwelling male non-smoker accounted for. There can be little doubt, however, that atmospheric pollution is otherwise noxious if not positively carcinogenic.

In 1950 Doll and Hill, on behalf of the Medical Research Council, conducted a survey of 1,357 patients with bronchial carcinoma in 20 London hospitals, and compared them with an equal number of 'controls' of the same ages. Smokers of 25 cigarettes or more a day comprised 25 per cent. of the cancer group, 13 per cent. of the other; only 7 men of the whole 1,357 cancer patients were non-smokers. This exhaustive and meticulously conducted inquiry suggested, in sum, that above the age of 45 the risk of contracting a carcinoma increased in simple proportion to the amount smoked, and was about 50 times greater in those who consumed 25 or more cigarettes a day for 20 years than in non-smokers. 9 per cent., or 1 such man out of every 11, can expect to die of the disease.

About the same time Wynder and Graham compared 600 bronchial carcinoma patients with similar controls: only 13 per cent. were non-smokers as opposed to 14.6 per cent. of the controls, and 51 per cent. smoked heavily (that is, 20 or more a day) compared to 19 per cent. in the control group. They also extracted cigarette tar with acetone and produced squamous carcinomas in 44.4 per cent. of experimental mice by painting their backs with the solution. Acetone alone caused no irritation to controls. In several cases two, and in one animal three, separate growths appeared; but the average time that all took to develop was about equal to half the lifetime of the mouse, a period equivalent to 30-odd years of smoking in a human.

Four carcinogens have now been identified in cigarette smoke, 3:4-benzpyrene and 1:2-benzanthracene greatly predominating, and both occurring also in industrial smokes. Also present are arsenic and radioactive potassium, but in such minute quantities that their effect is probably negligible.

Since the original work of Doll and Hill, and Wynder and Graham,

17 similar surveys have been made in a number of countries throughout the world, all with closely corresponding results. The American Cancer Society in 1952 recorded the life and smoking histories of no less than 188 000 men 11 870 of these have now (June 1957) died, 397 of lung cancers, the overall death rate being 68 times higher among the smokers. A survey by Doll and Hill of over 40 000 British doctors is under way and has already yielded similar evidence, the death rate from cancer of the lung being 40 times higher for heavy smokers than for non-smokers.

No significant difference has been found between those who do and those who do not—or say that they do not—inhalate perhaps because the latter aspirate the carcinogens without being aware of it. The incidence of the disease is, however, lower among pipe and cigar smokers.

There is no justification for the view that smoking merely determines the site of origin of a cancer which the victim would have got elsewhere had he not developed it in his lung for were this the case the death-rate among heavy smokers from cancers at other sites should be lower than in the non smoking group and this is not the case. On the contrary cancer of the larynx is ten times commoner among men than women in the United States, and heavy smoking greatly increases its incidence—an exact parallel to cancer of the lung.

In view of all this evidence we must therefore accept as proven

- 1 that the incidence and increase of bronchial carcinoma is related to cigarette smoking
2. that this varies directly with the amount smoked and the duration of the habit, the incubation period being about 20 years
- 3 that no satisfactory evidence so far exists of any other factors playing a significant part in the increase of recent years.

Out of any 1,000 men smoking more than 20 cigarettes a day 86 will probably die of lung cancer but among 1 000 non-smokers less than 5 will do so

As bronchial carcinoma does occur among non-smokers tobacco cannot be the sole cause and other causes for it must be sought. It has long been known that the Schneeberg and Joachimsthal miners, exposed to cobalt, bismuth, nickel and arsenic, as well as to considerable radioactivity suffered an incidence of between 40 per cent and 50 per cent. and chromate working in Germany and the United States also predisposes to the disease, as does nickel refining in this

country Asbestosis is associated with carcinoma, but, surprisingly, not silicosis, no significant difference being found between the incidence of lung cancer in miners and in the rest of the male population. Mineral irritants such as the foregoing clearly affect a very small minority.

The frequency with which chronic inflammatory lesions cause squamous metaplasia of the bronchial mucosa, converting the columnar ciliated cells into flat squames, might suggest them as predisposing factors to malignant change. No proof exists that this is so; but several times in my own experience I have seen squamous or anaplastic growths develop in the midst of areas of chronic bronchiectasis or emphysematous bullae; and they have also been described arising in bronchogenic cysts. Chronic bronchitis seems to bear some relation to carcinoma, but is itself markedly connected with smoking. There appears to be no link between influenza, such as the epidemic of 1918, and later

Pathology

All primary bronchial carcinomas arise in the bronchial mucosa and reproduce varying degrees of differentiation. They may arise in one or other of the lobes of the lung, or in the trachea which is usually involved in about 25 per cent distally, from periphery to centre. As they increase in size the greater the bronchial obstruction with all its consequences. There are three histological types, squamous-celled, and adenocarcinomas. The distinction between the whole, and between them and adenocarcinoma rate, the numbers of adenocarcinomas are constant. There are some grounds for regarding them as merely the anaplastic form of adenocarcinoma. They vary widely in their degree of differentiation. It is difficult to divide them neatly into types. There are differences of opinion between pathologists, and some placing squamous carcinoma in the category of 'giant-celled' or 'pleomorphic'

1. *Squamous-celled growths*

These are in general the most malignant; and make up .

are often stratified, and vary from polygonal to large flattened squames. The more differentiated they are the more frequent are cell nests and keratinization. The older the patient the more likely is his growth to be a squamous-celled one. Metastases tend to appear later, and the prognosis is consequently better. 75 per cent. of all five-year survivors of resections having originally had squamous growths. Because they are well differentiated and not very vascular, they tend to break down in the centre into chronic abscesses, and when this is seen radiologically the appearance is characteristic (X rays 49, 50, 55).

2. *Anaplastic carcinomas*

Oat-celled carcinomas are so called because they are composed of small, oval uniform cells, with big darkly-staining nuclei and scanty cytoplasm which resemble grains of oats. They are in other words undifferentiated, anaplastic and therefore much more malignant metastasizing early. At one time indeed they were thought to be sarcomas. They make up some 30 per cent. of the total and when bronchial carcinomas afflict people under the age of 45 as they frequently do they are almost certain to be undifferentiated, and the prognosis is bad.

3. *Adenocarcinomas*

Adenocarcinomas arise in the mucous glands of the bronchus and reproduce their elements, the cells being columnar, arranged in acini like a glandular structure, and often secreting mucus. They would appear to have a different origin from the other two varieties firstly because they have not increased with them and account only for about 5 per cent. of bronchial carcinomas and secondly because they are relatively much commoner in women, for their incidence is the same in both sexes. When the non-smoker gets a cancer it is most likely to be adenocarcinoma. They vary a great deal in malignancy some metastasizing early but some taking years to do so. They comprise many of the peripheral, circumscribed neoplasms in the lung and once they invade the pleural space disseminate throughout it so that seedlings of growth are found scattered all over the pleural surface, making identification of the primary impossible. Later these coalesce to form a continuous ragged sheet which is accompanied by profuse and recurrent pleural effusion eventually containing blood. It is this type of growth which is still sometimes diagnosed as endothelioma of the pleura.

Metastasis

Bronchial carcinomas metastasize in three ways, by the lymph channels, by the blood stream and more rarely, by the air passages themselves (X-ray 56). Peripheral growths tend on the whole to metastasize earlier than central ones. Lymphatic spread occurs first to interlobar and hilar glands; and thence upwards to the many mediastinal glands on the trachea, at its bifurcation and in the pulmonary ligament. Much of the lymph drainage of the lower lobe of the left lung passes to the carinal glands and thence across to the right paratracheal group, and, perhaps for this reason, the prognosis of growths in this lobe is less good than it is for neoplasms in other lobes. Later spread is up by the same chain into the supraclavicular fossae. Axillary lymphatic glands are very seldom involved in carcinoma of the lung

Malignant cells or emboli of growth reach the blood stream by the pulmonary veins and are therefore likely to lodge first in the brain. Particular care must be taken in handling a lung at operation not to squeeze the neoplasm so that emboli are detached. The liver is another common site for blood-borne metastases; and after it the bones, skin, suprarenals and other organs

Spread by the air passages is less usual, but growths not only extend by growing directly along them, or in their submucosal lymphatics, but sometimes groups of malignant cells are aspirated distal to the main neoplasm, giving rise to secondary deposits scattered throughout a lobe, every one being related to a bronchiole (X-ray 56), or cells may spill over into a dependent part of the lung or to the opposite side, or become implanted as seedlings in the main bronchi or trachea.

Symptoms

At present two out of every three cancers of the lung are inoperable by the time they reach a surgeon. Yet if it is diagnosed early and operated on promptly bronchial carcinoma is often a curable disease. The average time that elapses today between the first unmistakable symptom, and the arrival of the patient on the operating table which offers him his only chance of survival, is eight months—more than enough time for a growth to become inoperable, indeed a man's average expectation of life after diagnosis in untreated cases is—eight months. In spite of this, 40 per cent of patients in whom it is still possible to resect the lung can expect to be well two or more years after the operation. Where, then, does fatal delay take place? The following analysis of 135 case histories of men admitted to the

Thoracic Surgical department of the London Hospital during the sample year 1954 provides some answer

AVERAGE DELAY IN MONTHS

Thoracotomy Lobectomy Pneumonectomy
(*i.e.* inoperable)

Time between first symptom and reporting to doctor	1.0	1.2	2.0
Delay in doctor sending patient to Chest Clinic or local hospital	1.3	.9	1.2
Delay in Chest Clinic or local hospital forwarding patient to surgeon	2.0	5.5	4.0
Surgeon's waiting list	5	5	5
Admission to operation	5	7	.5
Total waiting time	<u>5.3</u>	<u>8.8</u>	<u>8.2</u>

It will be seen that patients report their symptoms to their general practitioners reasonably promptly and that they in turn send the patient on without much delay. Unfortunately however they do not decide for themselves 'This man probably has a cancer of his lung; his only hope is to reach a surgeon quickly' but instead, 'There's something wrong with his chest, let's send him to a physician!' Here average delays of between four and five and a half months take place. It is significant that those cases which eventually proved to be inoperable were kept waiting a much shorter time than those which were removable. In other words cachexia was recognized, but not early symptoms.

Some time has to be spent investigating and preparing patients but not too much time. In any case such investigations will almost certainly have to be repeated by the surgeon and are better carried out by him in the first place. Cancer of the lung should be regarded by everyone in contact with the patient as an acute surgical emergency. At any time the growth may invade structures which render it inoperable. At any hour of the day it may disseminate throughout the blood stream. In its treatment time is life.

The only way in which the prognosis of bronchial carcinoma can be improved is by prompt recognition of its early symptoms. Of these the earliest, and commonest, is

1 Cough

Unfortunately this is difficult to assess as so many men over 45

in this country have a 'winter' or 'smoker's' cough which is largely disregarded. But if, in such a man, a winter cough persists into the spring, or an unproductive cough becomes productive, or expectoration suddenly ceases, a chest X-ray must be taken. For growths which partially obstruct a bronchus promote infection distal to themselves, and hence cause purulent sputum, but if they block it altogether sputum stops. In men without a cough the mere presence of a neoplasm is irritative, and may give rise to one, masked but not cured by cough mixtures and linctus. The surgeon is powerless if a family doctor has not said to himself in time, 'This man's cough may mean a growth'.

2 *Pain*

It is unexpected that pain should be the next earliest common symptom, for it suggests the last stage of the disease and not the first; but most pain in the chest arises from the pleura, and although the chest wall may be directly invaded by a peripheral growth, much more often the pleural reaction is in response to segmental or lobar collapse following proximal obstruction of a bronchus by neoplasm. On admission to hospital at least 60 per cent of patients have X-ray evidence of pulmonary collapse of some kind (X-rays 3, 4, 5, 58). This 'pleurisy' is often transient, and if treated symptomatically, by a poultice and a pill, the warning which might have been in time, will be missed. Pleurisy in men of cancer age always demands a chest X-ray.

Sometimes a patient means even less than this by 'pain', implying rather 'discomfort', the inability (due to a solid tumour growing in the lung's elastic mesh) to take a full and satisfying inspiration.

3 *A febrile chest illness*

Variously described by the patient and sometimes, regrettably, by his doctor, as 'a touch of 'flu' or 'a patch of bronchopneumonia', this consists of pyrexia and malaise, often accompanied by a cough productive of some purulent sputum, and perhaps preceded by transient pleural pain. It is due to infection of the dammed-up secretions in a partially or completely obstructed bronchus. If penicillin is given, as it nearly always is under such circumstances, the fever and the symptoms will probably be suppressed, and the cancer causing them ignored. Even if an X-ray has been taken, the shadowing is likely to diminish and what is left is labelled 'unresolved pneumonia'. The term ought never to be used: it serves only to deceive.

the user. Pneumonia resolves, slowly sometimes, but progressively, and if it doesn't resolve it should not be called pneumonia.

In others the consequences of bronchial obstruction and infection are much more florid, and lead to lung abscess, lobar pneumonia or empyema. All lung abscesses and all empyemas, as well as any unresolved inflammatory lesions occurring in men over the age of 45 must be assumed to be due to cancer until the contrary is proved and in all bronchoscopy is imperative.

4 Haemoptysis

It will be observed that haemoptysis is by no means always the first symptom of bronchial carcinoma. If it were so more cases would be diagnosed early for the spitting up of blood is the one sufficiently dramatic and alarming sign to send a patient running to his doctor and for which his doctor will seek urgent advice. The haemoptysis of cancer of the lung consists of a bright streak of blood mixed with the sputum and repeated each morning for several successive days up to a week or more. This is so characteristic when it occurs that it is in itself almost diagnostic and no middle-aged man who has such a haemoptysis must escape bronchoscopy even if his chest X-ray reveals no abnormality of any kind.

5 Breathlessness

The next symptom in order of frequency is breathlessness because so many men of cancer age are already emphysematous, and the sudden collapse of a pulmonary lobe, or even segment, is enough to produce in them real shortness of breath. Partial obstruction of a bronchus may also cause a *persistent wheeze*. Rarely this is an early sign usually it is a late one heralding mediastinal obstruction.

6 Loss of weight

This is always of sinister significance but does not always spell inoperability. To expect it, or worse wait for it as confirmatory evidence of cancer, is to wait for death.

7 Other late symptoms

Other late symptoms all of them indications that the patient has passed beyond surgical aid, are *loss of appetite* and nausea, which almost always mean that secondary deposits have lodged in the liver, *hoarseness* due to malignant infiltration of the left recurrent laryngeal nerve, *difficulty in swallowing* caused by oesophageal involvement, the persistent *brachial plexus pain* and *Horner's syndrome* which

together constitute the Pancoast syndrome and indicate a tumour growing from the thoracic outlet, and the *cervical venous distention*, *swelling of the face* and eventual *stridor* of generalized mediastinal obstruction

The order in which the earlier symptoms have been given is that in which they most commonly occur. It is of course frequently the case that a patient first presents with haemoptysis, or with breathlessness, or even complaining of sudden loss of voice, or the very first symptom may be caused by ultimate dissemination a young architect, aged 32, apparently enjoying the best of health, complained to his doctor of a sudden sharp pain behind his left collar-bone. He had a pathological fracture of the first rib due to a metastasis, and both lung fields showed a 'snowstorm' of others

A well and pretty girl of 19 similarly had a pain under her left collar-bone. It was due to a secondary deposit from a hopelessly inoperable oat-celled carcinoma at the hilum of the left lung (X-ray 59)

Just as metastases cause local pain when lodged in bones, so do they give rise to a wide variety of neurological signs and symptoms when located, as they often are, in the brain Note that such symptoms, from either bone or brain, are unilateral and asymmetrical in distribution.

In many cases today lung growths are discovered by routine radiography unaccompanied by any symptoms whatever and the diagnosis rests on the radiological appearances alone

In others, preceding any of the commoner symptoms, or combining with one or more of them to complicate the clinical picture, the more unusual manifestations of bronchial carcinoma may be encountered

Unusual manifestations

(1) *Hypertrophic pulmonary osteoarthropathy* Although unusual this is certainly not rare, being seen in some 5–10 per cent of lung cancer patients It frequently gives rise to early, and characteristic, symptoms long predating the pulmonary ones, and failure to recognize these symptoms leads to much misdirected treatment Hypertrophic osteoarthropathy is generally, but by no means always, accompanied by clubbing of the fingers and toes, the clubbing sometimes appearing late in the syndrome or remaining inconspicuous. The first symptom is pain in the peripheral joints, the wrists and elbows, the ankles and knees This is aching in character and always worse at night when the patient gets warm in bed, so that it keeps

him awake. In many cases small effusions into the knee joints or elbows are seen and clubbing of the fingers and toes can usually be detected. In some it is gross in others virtually absent. All these signs and symptoms, unlike those due to secondary deposits are *bilateral* and *symmetrical*. The pain is always the symptom complained of but it is frequently disregarded, or diagnosed as rheumatism or arthritis and its grave significance ignored. Although clubbing is seen in congenital heart disease and in chronic suppurative conditions of the lung, in these it is not usually associated with joint pains whereas it is never seen at all in uncomplicated pulmonary tuberculosis, and this fact is of great diagnostic value.

In hypertrophic osteoarthropathy there is a deposit of subperiosteal new bone related to the epiphyseal ends of the long bones and at the muscular insertions into their shafts (X ray 60). Accompanying this is a tendency to osteoporosis of cancellous bone and in the finger ends (causing clubbing) there is increase in the fibro-elastic and fatty tissue of the nail beds, with the formation of new capillaries. All these changes are explained by increased peripheral blood flow, which results in overnutrition of the parts.

X rays of the long bones show subperiosteal bone deposition when the condition has been present for some time and although this evidence is conclusive a history of peripheral joint pain of sudden onset and persistent course, which is worse at night, and may be combined with early clubbing or effusions is equally so and demands urgent investigation of the lungs. The severity of the symptoms bears no relation to the size of the growth, and indeed very troublesome and long-standing pain is often associated with a very small peripheral neoplasm.

Removal of the growth by lobectomy or pneumonectomy immediately abolishes all the joint pain, which never returns, even if the patient subsequently develops metastases. This is because the increased blood flow through the limbs which causes the bone and soft tissue changes is controlled by the lesion in the lung through the fibres of the vagus nerve of the same side. The act of resection cuts these vagal fibres and at once diminishes the flow. Similarly, should a carcinomatous lung prove inoperable the troublesome pain which is frequently the patient's most bitter complaint, can be instantaneously relieved by performing a simple vagotomy just below the recurrent laryngeal branch, on the affected side. Within 24 hours nail-bed swelling is also diminished and effusions begin to absorb.

A business man of 50 suffering from severe joint pains was treated for a year in a physiotherapy department for rheumatism

A mass X-ray of his chest revealed a small round lesion in the upper lobe of his left lung. It was a primary carcinoma, and the morning after lobectomy all his pains vanished. They never returned.

A man of 63 had been under periodic X-ray observation for an undoubted tuberculous focus at his right apex for many years. In his most recent film there was some increased opacity near the lesion, and although it was thought probably to be due to an extension of senile tuberculosis, and he was said to have no symptoms referable to it, he was referred for surgical advice. I asked him 'Have you recently noticed rheumatic pains?' 'Yes,' he said, 'in my knees and wrists; and a little in elbows and ankles too.' Although he had no clubbing, the differential diagnosis between tuberculosis and cancer was not now in doubt and he was subsequently successfully operated upon.

A man of 45 had first swelling and then pain in his ankles. A month later his knees were swollen and painful, and after a further month his wrists and elbows were affected. He had gross clubbing of fingers and toes, and substantial effusions in both elbows, knees and ankles. Radiography confirmed new subperiosteal bone deposits, and showed a small opacity in the upper lobe of his left lung. Squamous carcinoma cells were present in his sputum. He agreed to a two-stage operation. At the first stage a small intercostal incision was made, the presence of an operable carcinoma in the upper lobe confirmed, and the left vagus nerve isolated, picked up just below its recurrent laryngeal branch, and cut. Next morning he was asked if he felt any different. 'Goodness me, yes,' he said, 'all my pains are gone; and look—I can bend my knees for the first time in weeks.' In 48 hours the effusions in the knees and ankles had almost disappeared, and the skin at the base of his nails was beginning to wrinkle. Within a few days his chest was re-opened and a lobectomy performed. The second operation made no difference, all his symptoms having already disappeared.

(11) *Carcinomatous neuropathy* This is much rarer than hypertrophic osteoarthropathy, having an incidence of about 2 per cent. It also differs radically from it, although it shares the important feature of being bilateral in its manifestations, and is thus easily distinguishable from neurological changes due to secondary deposits in the brain or spinal cord. Although it is usually concurrent with a bronchial carcinoma, it is not tightly linked to it by a neural reflex as is osteoarthropathy, and may long precede a growth, or appear only some time after one has been removed. The symptoms may spontaneously remit, but they cannot be relied upon to do so after



49

X-ray 49. Abscess cavity with fluid level in apex of right lower lobe. Although this is a common site for aspiration because it can only be a breaking-down squamous carcinoma. Note complete irregularity of wall. The appearance is utterly unlike that seen in X-rays 51 or 52 or 4.

X-ray 50. A typical breaking-down squamous-celled carcinoma in the upper lobe of the right lung. This is not only the commonest form of malignant abscess—it is the commonest type of lung abscess. Such a cavity in the chest X-ray of a man of cancer age demands a diagnosis of bronchial carcinoma.



50



51

X-ray 51. Abscess distal to bronchial obstruction caused by growth in left lower lobe. It is exactly like an ordinary aspiration abscess. As fluid level is present obstruction cannot be complete, so abscess may melt away after antibiotics. If patient is not bronchoscoped growth will be missed. Compare X-rays 23 and 49.

X-ray 52. A lateral view of the same malignant abscess seen in X-ray 51.



52



53

X-ray 53 An aspiration abscess in the apical segment of the left lower lobe. The patient expectorated stinking pus and the abscess very rapidly resolved with antibiotics. Now see X-ray 54



54

X-ray 54 Abscess seen in X-ray 53 has completely disappeared. Note, however, small opacity in right upper zone (level of back end of 7th rib). This is an oat-celled carcinoma. A necrotic fragment from it was aspirated into left lung causing 'spill-over' abscess. Growth is just visible in X-ray 53



55

X-ray 55 A tomogram of a breaking-down squamous carcinoma in the left upper lobe. Although this growth does not contain a fluid level of pus the irregularity of its wall is apparent, and confirms the diagnosis. It was perfectly operable. (Compare with X-rays 49 and 51)



56

X-ray 56 A carcinoma of the right upper lobe with air-borne secondary deposits distal to it in the upper lobe. None was present elsewhere in the body or the lung and the disease was extirpated by right pneumonectomy

a resection, and in fact seldom do. The cancer and the neuropathy would thus seem to share a common origin, perhaps in some metabolic change in the body predisposing to both, and whereas osteoarthropathy is a manifestation of bronchial carcinoma, the various forms of neuropathy are sometimes seen in connection with carcinomas of the breast or ovary although they are commoner with those of the lung. There are three forms of neuropathy, of which the first is the most usual.

(a) There is a generalized loss of muscle tone in the extremities followed by weakness and wasting, diminution of reflexes, much loss of weight and finally atrophic paralysis. In most the legs are first affected, but in some the symptoms resemble bulbar palsy and there is dysarthria and dysphagia. Although this syndrome much resembles myasthenia gravis the use of neostigmine gives no relief.

(b) The predominant disturbance is sensory with tingling, numbness, cramp or shooting pains in the extremities, tenderness in the muscles, sensory loss, and finally weakness and muscle wasting as in the first group. The symptoms are therefore more like peripheral polyneuritis, subacute combined degeneration or disseminated sclerosis.

In both these groups degeneration of the posterior columns in the cord, of posterior root ganglia, or of the peripheral nerves themselves has been demonstrated and it appears to affect any point along the neuromuscular path, so that both syndromes are aspects of the same disease, sensory symptoms predominating for a time in the one, motor in the other. Remissions, some complete, are a feature of both and death, when it comes, is due not to the neurological disorder but to the growth. The disorder has only once or twice been described in connection with carcinomas other than bronchial so that symptoms of atypical and unexplained polyneuritis, disseminated sclerosis or myopathy demand radiological examination of the chest. In most patients growth and neuropathy will coexist but the strangely capricious nature of their relationship is exemplified in these two case-histories.

A man in his thirties developed what seemed to be a carcinomatous neuropathy with sensory and motor changes but full investigation of his chest, including bronchoscopy proved negative. He was kept under observation, and in the course of some nine months largely recovered. More than a year after his first symptoms he presented with secondary deposits of a bronchial oat-celled carcinoma.

A publican of 51 who had no neurological symptoms of any kind, underwent a successful left lower lobectomy for an oat-celled

carcinoma. Two metastases were present in regional lymph glands removed at the same time and the prognosis seemed poor, so I was not at first surprised when a year later he began to lose weight and complained of weakness in his legs. Difficulty in walking increased and in six months he lost two stone in weight, his left leg became almost flail-like and he found it hard to stand upright. Later both shoulders were affected. There was much symmetrical muscle wasting but no sensory changes. After some months in this state he began to improve and to regain weight. It is now six years since his operation, he is alive and well, and apart from slight weakness in one leg has completely recovered from his neuropathy.

(c) The third type of neurological disturbance is one of subacute cerebellar degeneration with marked ataxia, dysarthria, diplopia, nystagmus and a reeling gait, ending in dementia. There is marked loss of the Purkinje cells in the cerebellar cortex, as well as degeneration in some of the spinal tracts, and the condition is associated with other carcinomas than those of the bronchus. It is progressive and never remits.

(iii) *Spontaneous pneumothorax* I have several times seen carcinoma of the lung presenting by spontaneous pneumothorax in patients with emphysematous lungs. The growth interferes with the air-flow to a group of alveoli, tension develops, the alveolar walls are ruptured, a bleb forms, and finally bursts into the pleural cavity causing pain and sudden dyspnoea (X-ray 61).

(iv) *Ballooning cyst of the lung*. This is a variation of the foregoing, a bulla increasing in size owing to bronchial obstruction. The presenting symptom is shortness of breath (X-ray 36).

(v) *Persistent wheeze*. This is common in the presence of mediastinal obstruction, but rarely complained of by patients with early, operable growths. The following history is packed with valuable lessons for all who treat disease of the chest.

An intelligent schoolmaster went to his general practitioner and said, 'Doctor, I have a wheeze in my chest just *there*, and it doesn't go away after I've coughed.' He pointed to the front of his left chest in the region of the third interspace. His doctor listened, confirmed that there was indeed a persistent rhonchus to be heard—that is a rhonchus which is unchanged by coughing and therefore denotes an incomplete organic bronchial stricture—and referred him to a consultant chest physician at a special chest hospital. The physician X-rayed him, tested his sputum and bronchoscope him with negative results. Finally bronchography of the left lung was carried out, and, after he and the radiologist had passed the broncho-

grams as normal, he sent the man home again. In time the wheeze disappeared, and one year later he was referred with pain in the chest and loss of weight. He had now an obvious carcinoma in the anterior segment of his left upper lobe. When the bronchograms of a year before were obtained, the antero-posterior views seemed perfectly normal but the lateral views at once showed that all the bronchial segments had filled with dye—save the anterior segment of the upper lobe (X ray 30). This finding, though negative, is just as significant as a positive abnormality and it dramatically confirmed both the patient's history and his signs on physical examination. On it, as it turned out, hung his life.

(vi) *Obstructive emphysema* If a neoplasm in a main bronchus partially occludes it, so that air can enter the lung as the bronchus expands on inspiration but cannot escape again on expiration, progressive breathlessness due to obstructive emphysema results. X rays of the chest taken, as is usual, on inspiration may look quite normal but if the patient expires air leaves the unobstructed lung only so that the mediastinum is displaced to the normal side, while on the side of the growth the diaphragm is unable to rise and the lung is more translucent (X ray 62). Only bronchoscopy will reveal the neoplasm.

(vii) *Auricular fibrillation*. This may be present in elderly patients and have no connection with a carcinoma but sometimes it is due to direct invasion of the heart by growth and is only one of several reasons why patients with cancer of the lung find their way first to cardiological departments. In some it is because they are short of breath in others there is precordial pain, in others there is fullness of the neck veins from mediastinal obstruction and yet others have cardiac irregularity.

(viii) *Expectoration of growth* Carcinoma cells are present in the sputum of the majority of patients. A few however spit up macroscopic fragments of growth. A patient of mine aged 60 who was otherwise quite well and symptomless, had a sudden attack of coughing during which he spat up a fleshy mass about the size of his thumbnail. This he carefully preserved and took to his doctor who sent it to a pathologist for section. It proved to be oat-celled carcinoma. Radiologically his lung fields were completely clear. Bronchoscopy was normal. Detailed tomography of both lungs revealed only a small lung cyst on the left which did not justify exploration. Where had the growth come from? In the end I sent him away for a time, and further X rays in a month showed a minute opacity in the right upper lobe which was then resected. He remains alive and well.

Diagnosis

Although cancer of the lung may be found at any age and in either sex, it is so common in men between the ages of 45 and 65 that should a suspicion, either clinical or radiological, fall upon such a patient it must be pursued to a conclusion

History

In the course of a detailed history of his symptoms the patient should also be asked, 'Do you smoke?' If his answer is 'No', he is then asked, 'Have you ever smoked?' In many cases it will be found that he gave up smoking heavily with the onset of his symptoms. It is also important to find out whether he was able to work, and to walk, as fast as other men of his age before his illness began, for inability to do so suggests emphysema of the other lung, or heart disease, which may render surgery hazardous. Can he still work? Can he run for a 'bus, or climb a flight of stairs without halting? A long history referable to lung cancer is not in itself forbidding unless it is accompanied by marked deterioration; for it suggests the low malignancy of a squamous growth which may remain perfectly removable even after a year or more of growth. On the other hand a short history in an otherwise robust young man probably means an anaplastic growth of doubtful operability. Inquiry must always be made about the appetite, for loss of it suggests hepatic metastasis; about swallowing, for dysphagia means inoperability, as does hoarseness; about 'rheumatism' in the peripheral joints, for the patient is unlikely to associate it with his lung, and about possible neurological disorders.

Physical examination

This must include the whole body, not just the thorax.

Does the patient show any signs now of breathlessness at rest, or on climbing a flight of stairs? Is there cyanosis of his face or mucous membranes? Is his skin slack, suggesting loss of weight, and are there secondary deposits to be felt in it? Enlarged glands must be sought in the supraclavicular fossae and elsewhere, the liver palpated, a rectal examination done. Pain in the limbs, backache or 'sciatica' must be fully investigated lest metastases are responsible. Neurological lesions are carefully excluded, for they may be due either to secondaries or to a neuropathy. What is the sputum like? Is it profuse and mucoid and frothy suggesting chronic bronchitis or heart failure, or does it contain pus, or blood, or both?

Physical examination of the chest may elicit a great variety of signs diminished movement on the affected side displacement of the trachea and apex beat towards the lesion by collapse or away from it by an effusion dullness due to fluid or a mass resonance due to emphysema or a pneumothorax diminished air entry, bronchial breathing, absent breath sounds a persistent ronchus or the rattling râles accompanying infection. But it may be absolutely negative and even the most thorough physical examination in a suspected case of bronchial carcinoma is not enough The most subtle percussionist can overlook gross lesions in the lung

Radiological appearances

No examination of the chest is complete without good postero-anterior and lateral X ray films and these may show a wide variety of abnormal shadows In 60 per cent. of cases they are caused by collapse of a lobe (see Fig. 6) or a segment, or of the whole lung (X rays 3 4 5 58, 63) The larger the area collapsed the greater is the tendency for displacement of other structures towards it and for added translucency in the adjacent lung due to compensatory emphysema. Usually the airless, opaque segment is, from one or other aspect, triangular and has a concave margin In another 10 per cent. or more an abscess of some kind is visible containing a fluid level, most often due to necrosis in a squamous growth, when its walls are seen to be thick and irregular (X rays 49 50 55) or, if it lies distal to a bronchial obstruction, or is caused by spill-over of pus or necrotic material from one part of the lung to another exactly resembling an ordinary pyogenic lung abscess (X rays 51 52, 53) Together these three varieties of abscess are the commonest abscesses found in the lung so that a lung abscess of any kind discovered in a man over 45 must always be regarded as malignant until its innocence is proved. They are discussed in greater detail in the chapter on 'Lung Abscess'

Growths which have not yet caused collapse, or broken down to form an abscess cavity are typically irregular, with ill-defined edges often situated near the hilum and flaring out into normal tissue (X ray 56) Such appearances can by no means be relied upon rapidly-growing anaplastic carcinomas usually have a smooth lobulated edge very like that of innocent tumours (X ray 65) whereas some cancers are compact and discrete and situated so peripherally that they resemble rather a tuberculoma or hamartoma (X rays 54 57).

It is in particular these symptomless small round lesions or coin

shadows' which are increasingly detected by mass radiography and, as they are far beyond bronchoscopic vision, they pose difficult diagnostic problems (X-ray 54) Out of 50 consecutive such lesions, in patients of all age groups, sent to me from mass X-ray units, no less than 19, or 38 per cent., proved on removal to be early primary carcinomas, 23, or 46 per cent., were tuberculomas, and the remainder were an assortment of hamartomas, cysts and small inspissated abscesses The chief distinction has to be made therefore between cancer and tuberculosis, together constituting 84 per cent of the whole, and it is noteworthy that in either case the right treatment of this kind of lesion is excision Some points of difference are seen in the following table

<i>Carcinoma</i>	<i>Tuberculoma</i>
19 cases (38%) .	23 cases (46%)
All male	14 male, 9 female
Average age 51 .	Average age 36
16% were <i>under</i> 45	30% were <i>over</i> 45
63% were anterior .	58% were posterior
Malignant cells in sputum of 4 (21%)	Direct or culture positive in 8 (33½%)

Although the chances of an isolated lesion being malignant are much higher in patients over 45, the youngest in this group was only 35 The majority of tuberculomas are found in the back half of a lateral X-ray because they are most often situated just below the apex of one or other lobe, so that anterior shadows are additionally suspect It will be seen, however, that this distinction reflects only a trend, and there is plenty of overlap in both directions Sputum tests may help, but three of my cancer patients had evidence of old tuberculosis and one had a positive sputum

It is indefensible to sit and watch such shadows to see if they are increasing in size failure to grow is no guarantee of innocence Many examples are on record of these lesions being observed for several years under the delusion that they were tuberculomas before the inevitable explosion occurred A man of 43 was discharged from the army in 1949 after X-rays showed a 'small round tuberculous lesion' in his middle lobe He was X-rayed regularly and no change occurred. In 1952 he was referred to a medical colleague who advised the removal of his 'tuberculoma' and sent him to me The patient refused operation on the grounds that he was symptomless and well, and that the shadow had not altered in three years This seemed reasonable and I agreed to keep him under further observation In

1953 the edge of the lesion showed some very slight changes. I insisted on removing it. It proved to be an adenocarcinoma, and had remained virtually the same size for 4 documented years.

To watch such lesions is like sitting on a powder keg (X ray 59). It is seldom possible to be certain of the identity of an isolated round lesion in the lung until it is removed, and even should it then prove to be a tuberculoma excision is the best treatment for it. Any other policy is a blind gamble with the patient's life, particularly if he is over 45. The only right rule for the management of such a shadow is quite simple: *when in doubt, take it out*.

X ray screening helps to determine the mobility of a shadow, its relationship to the heart and mediastinum, and to swallowing. The patient must be asked to sniff while the excursion of both sides of his diaphragm is watched; normally both jerk downwards; if one side instead flaps upwards, this paradoxical movement is a certain sign that phrenic paralysis due to infiltration of the nerve by growth, has occurred and there is little or no chance of removal. Similarly if a barium swallow shows oesophageal distortion inoperability is certain. Bronchograms are sometimes useful in demonstrating a partial bronchial obstruction in early lesions beyond bronchoscopic view. Dionosil, which clears quickly, should be used.

Tomography is frequently valuable in affording a clearer picture of obscure lesions, in demonstrating central cavitation in them, and in establishing their exact relationship to nearby structures (X ray 55). Cavitation in a doubtful shadow is strong evidence of malignancy.

Sputum examination

The sputum must always be examined for predominant organisms (whose sensitivity to antibiotics is determined) for tubercle bacilli and for carcinoma cells. Staining the sputum for this last investigation is technically easy by the Dudgeon, Papanicolaou, or Perrin dyes, but the actual recognition of the malignant cells calls for long training. It should not be attempted within ten days of bronchoscopy for instrumentation causes the mucosa to shed cells which are easily mistaken for those of squamous carcinoma. Negative findings are of little significance, but positive ones provide valuable confirmatory evidence in cases where the lesion is beyond bronchoscopic range. At the London Hospital positive cell reports are obtained in about 65 per cent. of patients.

Pleural fluid

Pleural fluid, if any is present, should likewise be searched for

malignant cells though they are difficult to identify in it and the investigation is seldom conclusive

Bronchoscopy

Bronchoscopy must never be omitted when cancer of the lung is suspected, even when the chest X-ray is quite clear. Emphysema and high blood pressure are very common causes of haemoptysis in middle-aged men, and are often found together: but failure to bronchoscope such a patient who has coughed up blood is culpable negligence.

The objects of bronchoscopy are:

1. to establish the diagnosis,
2. to determine technical operability.

It is therefore much better carried out by the thoracic surgeon who is himself to operate, rather than by a physician or some other third party whose only interest is in diagnosis.

The procedure is a simple one, and can be carried out under local anaesthesia, in the out-patient department or the consulting room (For a full description of the technique see the chapter on 'Bronchoscopy'.) It provides the only direct means of clinching the diagnosis with a biopsy

As 75 per cent. of growths arise in major bronchi it is possible in the majority of cases (70 per cent.) either to see them directly and to remove a fragment for histological examination, or at least to deduce their presence with fair certainty from the rigidity of a bronchus, from bulging of its wall, or from a stenosis

In the remaining 30 per cent., the growth lies beyond bronchoscopic vision, either because it is peripheral, or is situated in an upper lobe segment which cannot be seen through the right-angled telescope. In short, negative bronchoscopic findings mean only that the operator is unable to see a growth, not that there is no growth.

Bronchoscopy is indispensable in assessing operability. Lagging, or complete paralysis of a vocal cord (almost always the left), indicates that the recurrent laryngeal nerve is invaded as it lies on the mediastinum. The tracheal wall itself may be eroded; or the carina widened by enlarged mediastinal glands; or growth may extend so far up one of the main bronchi that no room is left to amputate it from the trachea. In all these cases removal of the lung is impossible.

Blood count

A blood count provides evidence of secondary anaemia, and the

worse this is, the worse usually is the prognosis. Quite often breathlessness attributed to the neoplasm will be found to be due to anaemia, and is relieved in large measure by transfusion. Leucocytosis suggests infection either in the growth itself or beyond the obstruction it causes in a bronchus.

The erythrocyte sedimentation rate

This is almost always raised in cancer of the lung and in the differentiation of a small carcinoma from a tuberculoma, a raised sedimentation rate favours carcinoma.

If all these aids to diagnosis prove inconclusive, and doubt remains as to the nature of an abnormal shadow in the chest X ray of a man aged 45 or more the shadow must be assumed to be due to cancer until its innocence is proved. For not more than a fortnight the effect of antibiotics may be tried. Inflammatory lesions will change in this time and if the shadow diminishes progressively in size, therapy is continued for as long as there is a positive response. But if clearing ceases, or a residuum remains, no further time ought to be wasted. Many carcinomas are associated with lung infection, in which most of the radiological opacity is due to pus or to consolidation, not to growth itself and these shadows often clear considerably but not completely with antibiotic therapy.

In a few exceptional circumstances it is justifiable to watch a lesion, for short periods only and under constant supervision, to see if its outline alters. Earlier X rays, taken at other hospitals in past years, are becoming more frequently obtainable and often provide conclusive evidence about the nature of symptomless shadows.

Attempts at needle biopsy, or aspiration of a mass through the chest wall, are to be heartily condemned. If they do not spread growth, they are very likely to spread infection into the pleural cavity.

The next logical step in diagnosis, where reasonable doubt still exists, is *thoracotomy*.

Selection of patients for surgery

In the treatment of lung cancer only surgical removal holds out good hope of cure. No candidate for surgery therefore should be rejected for any reason other than the presence of one of the signs of inoperability already mentioned and summarized below or unless he clearly cannot tolerate the removal of the affected lung. When faced with an unpromising prospect, the surgeon should say to himself: "If this were my lung would I like an effort made to take it out?"

The following are the absolute contra-indications to surgery.

1. *Distant metastases*, either palpable, or evidenced by local pain and tenderness; or unilateral nerve lesions, or anorexia and nausea
2. *Mediastinal obstruction*, with distention of neck veins, swelling of the face, and stridor
3. *Pancoast syndrome*, with brachial plexus pain, and Horner's syndrome.
4. *Recurrent laryngeal palsy*, causing hoarseness
5. *Phrenic paralysis*, causing paradoxical movement of one half of the diaphragm.
6. *Oesophageal involvement*, causing dysphagia.
7. *Blood-stained pleural effusion*, due to dissemination of growth over the pleura.
8. *Erosion of vertebrae*, demonstrated by X-ray.
9. *Tracheal involvement*, seen on bronchoscopy
10. *Carinal widening*, seen on bronchoscopy
11. *Growth extending up to the carina*, seen on bronchoscopy

The question of whether a patient will tolerate a pneumonectomy is best decided on clinical, not bronchspirometric grounds, and never on an estimation of vital capacity which is in no way a measure of pulmonary function. Elaborate laboratory tests and blood gas analyses never provide evidence which cannot be better got by watching a patient walk upstairs. Where doubt exists, the patient ought to be given the benefit of it, and it is often helpful to see how well his colour is maintained on 20 per cent oxygen after the intrabronchial anaesthetic balloon has blocked off his affected lung. It must also be remembered that a patient with a collapsed, or otherwise functionless, lung will be less breathless after the pulmonary vessels supplying it have been ligated and the blood they carry diverted to aerating tissue. A past history of coronary occlusion clearly makes a major operation more dangerous, as do also much emphysema or chronic bronchitis; but none of these things in itself forbids surgery. A considerable degree of hypertension is, on the whole, well tolerated by pneumonectomy patients. Any suggestion of congestive heart failure on the other hand calls for rejection. A growth demanding right pneumonectomy is always much more serious than one on the left side, for the right lung is the bigger in volume (having three lobes to the left's two), and its removal is therefore not tolerated so well.

Age in general is no bar, but a patient over 70 has to be in particularly good condition before he is acceptable. Below this age lung resection can usually be carried out with a very low mortality,

although in borderline patients who are already short of breath every effort should be made to remove the neoplasm by lobectomy rather than by pneumonectomy

Forbidding X ray appearances never justify rejecting a patient, and a long history implying low malignancy encourages determination rather than the reverse. Invasion of the chest wall, providing that the thoracic outlet and the vertebrae are spared, by no means precludes surgery and the involved ribs and intercostal muscles are resected *en bloc* with the lung, the gap in the chest wall being filled by the scapula, or by polythene sheeting sewn over it (X rays 66, 67, 68). If a pneumonectomy has been performed this is unnecessary, for the operation can be completed with a thoracoplasty. In the same way portions of the diaphragm or of the pericardium are easily excised, and the defects closed by approximation of the edges if they are small, or by floss-silk or plastic repairs if large.

Although a blood-stained pleural effusion contra indicates operation, a clear effusion or an empyema do not. Small effusions accompany collapse of a lobe and the growth causing this may be perfectly operable but if the effusion is massive and soon recurs after aspiration, it is more likely to be due to an adenocarcinoma spreading widely over the pleura. Thoracotomy will probably be necessary to establish this diagnosis and in early cases one can still sometimes strip off the pleural layers studded with nodules of growth so that a great deal of the neoplasm is removed and the lung adheres to the chest wall preventing further effusion. I have one such patient whose pleura contained numerous plaques of adenocarcinoma accompanied by massive effusion and who is very well indeed and quite free of effusion more than three years after such a pleurectomy.

Empyema frequently occurs secondary to infection in a collapsed lung (X ray 45) though it can also be caused by necrotic growth breaking down into the pleural cavity. In either case the growth may remain operable, and the lung is removed together with the whole of the pleura and the empyema.

Surgical palliation is often indicated in patients who have no chance of permanent cure. Whenever bronchial obstruction results in distal lung infection the patient is ill, fevered and toxic and is greatly benefited by the removal of the bag of pus which the lung has become even if the growth has already extended into the mediastinum. A high temperature is no contra indication to surgery in these cases, because once the infected lung or pleura is removed the fever ceases.

Growths which are threatening to invade the chest wall likewise

call for palliative removal since they are bound in time to cause severe and intractable pain (X-ray 66)

Surgical Treatment

The treatment of choice is prompt pulmonary resection in all who are technically operable; and there is an excellent chance of cure in about 40 per cent of such patients

Preparation 80 per cent. of the candidates for surgery are men between the ages of 55 and 65, most of them life-long heavy smokers. Many therefore are mildly bronchitic and emphysematous, and it is of special importance to fit them as best one can to sustain the loss of a lung or a lobe. In particular every effort must be made by concentrated breathing exercises to increase costal mobility and to secure maximum diaphragmatic movement. The efficiency of the exercises given is measured by the increase of vital capacity resulting from them, and this is the chief purpose of vital capacity estimations.

Postural drainage is often useful in draining the affected lung if bronchial obstruction in it is not complete, or if an abscess is present, and antibiotics are used in any case to suppress associated infection. They should be given for at least a week before, and continued for ten to twelve days afterwards, to cover the operation itself, but persistent preoperative fever is not a good reason for surgical delay.

Bronchospasm is relieved as far as possible by anti-spasmodics such as aminophylline, ephedrine and isophrenaline sulphate; and these are more effective when given in combination, by inhalation, by mouth, and by injection. Sometimes the removal of the growth is accompanied by spontaneous remission of bronchospasm on the opposite side, suggesting a reflex influence.

The haemoglobin percentage is raised by a blood transfusion (given at least four or five days before operation) to a level of not less than 80 per cent, a further minimum of 2 or 3 pints being needed during the operation itself. Much of a patient's dyspnoea may be due to anaemia.

The operation. The techniques for removal of part or all of the lung are fully described in the chapter on 'Pulmonary Resection'. The nature of the operation, whether it is to be a pneumonectomy or lobectomy, whether an intra- or extra-pericardial ligation of the vessels is to be carried out, and whether or not a block dissection of all accessible lymphoid tissue, subpleural fat and areolar tissue of the mediastinum is to be performed is decided strictly upon the circumstances, the age and state of the patient, the character and situa-

tion of the growth and not by any preconceived doctrine. It is wrong to remove a man's whole lung, especially on the right side, if lobectomy yields just as good results. There is no point in ridding a man of his cancer only to have him die of lack of breath. It is wrong to open the pericardium widely and expose an elderly patient to greatly increased risk of atrial fibrillation on account of an early neoplasm the prognosis of which is not altered one whit thereby. It is wrong to subject a frail and ill subject to lengthy and laborious block dissection exposing the heart and mediastinal structures for perhaps two or three hours, when an operation less radical in theory but as efficacious in practice lasts three-quarters of an hour. It is wrong to dethrone individual clinical judgement in the name of general principle paying the price of a raised death rate without yielding the dividend of more 5-year survivors. On the other hand the decision to perform as scrupulous a block dissection of the lymphatic systems of the mediastinum as possible is sometimes called for but this can never be more than an approximation to radical clearance.

The lung is one organ in two halves and its lymphatic drainage cannot be extirpated by operations confined to one pleural cavity; the lymphatics from the lower half of the left lung, for example, drain across to the right paratracheal chain. In addition the vital character of the complex structures which partially divide the two halves of the lung forbid effective clearance. No one advocates routine removal of the left recurrent laryngeal nerve either from the left or from the right sides or interference with the thoracic duct, which communicates with the mediastinal systems nor is any satisfactory clearance of the adventitia of the great vessels or of the coats of the oesophagus practicable. By all means let us be as thorough as possible in the removal of involved tissues but do not let us delude ourselves as to the anatomical completeness of such operations.

Postoperative Care

During the first week this is the same as the postoperative management of any patient after lobectomy or pneumonectomy and is described in detail in the chapter on Pulmonary Resection.

The patients on the whole are older than most, and consequently are more subject to cardiac complications, in particular atrial fibrillation especially if the pericardium has been opened during operation. Often the irregularity is transient, but should it persist prompt digitalization with intravenous digoxin is indicated. Some measure of congestive heart failure is not uncommon, and is aggravated by an

excess of fluid in the empty pleural space even when this is not accompanied by mediastinal displacement. The accumulation of frothy mucus in the bronchi under these circumstances should always be relieved by bronchoscopic aspiration, and this is frequently sufficient to turn the cardiac corner.

Longer bed rest is of course necessary for such people; but for most others early ambulation has great advantages, and the well patient should certainly be encouraged to get up on his second or third postoperative day. The mere fact of getting up so soon after an operation which he feared would end his active life gives the patient great encouragement, lifts his spirits, and speeds his convalescence. He is usually ready to go home or for a quiet holiday in less than three weeks.

If a pneumonectomy has been performed, treatment thereafter depends upon whether or not the empty pleural space fills up sufficiently with serum to keep the mediastinum central (X-rays 9, 10). In most cases it does so, but in some it is necessary to readjust the intrapleural pressures once a month with an artificial pneumothorax apparatus, and patients attend the out-patient clinic for this to be done. About 80 per cent of patients who are going to develop recurrence or metastasis do so within the first year after resection; and 98 per cent do so within the first twenty-four months. If a patient is well, without evidence of recurrence, and his weight is fully maintained at the end of two years, it is unusual for him to die of recurrence later. Should he still be in need of air-refills at the end of two years to keep his mediastinum central, he is then offered the alternative of a lateral thoracoplasty (X-ray 68).

The ability to work depends not so much upon the age of the patient as upon the condition of his remaining lung, and his psychological adjustment to his disability. All men under pensionable age are encouraged to return to useful work as soon as they are fit to do so, and the great majority find this possible within three months of their operation.

Results

Among 222 patients operated upon by me at one hospital up to and including 1953, 76 (or 34 per cent of the total) proved to be inoperable after thoracotomy; and most of these were dead within a few months, though some lingered on for as long as a year, and three lived over two years but were clearly going downhill by the end of the second. The average survival of 52 patients who subsequently died after resections was 10½ months.

109 pneumonectomies were performed and 36 lobectomies. Of the pneumonectomy patients 42, or 38.4 per cent, remain alive and well and of those who had a lobectomy 16, or 44.4 per cent. In one woman a pedunculated, wholly intrabronchial squamous carcinoma was removed by bronchotomy alone, five years ago and she is still very well. The overall survival rate, therefore, three years and more after resection is just over 40 per cent.

It will be noted that in spite of the much less radical excision involved by lobectomy the survival rate is actually higher than that after pneumonectomy this reflects largely the much earlier type of growth for which lobectomy is suitable.

Following the 222 operations 10 patients died while in hospital an overall mortality of 4.5 per cent. or if resectable cases alone are considered, 6.8 per cent. The most frequent cause of death was heart failure.

Patients undergoing resection varied in age from 19 to 74 but the great majority were between 55 and 68.

No patient was rejected for surgery unless one of the specific contra-indications to it already detailed was present and many purely palliative operations were deliberately undertaken in order to render the patient's remaining life more comfortable, but without any hope of cure.

Palliation

The treatment of any patient with inoperable cancer is difficult, but this adds to the responsibility of all concerned to see that it is conducted efficiently and with humanity. In every case there is *something* to be done, and in many a great deal, to alleviate symptoms and prolong tolerable life. Patients can be taught to live with inoperable carcinoma as they do with diabetes and other incurable diseases.

There are very few people strong enough to bear the burden of knowing the plain facts about their condition they should be told that they have a partial obstruction of a bronchial tube or a chronic infection of the lung or if they press for a specific label squamous metaplasia and that for all these things excellent non-operative treatment is available. This is the truth but not too much of the truth. Terms like carcinoma 'tumour' or growth must be avoided. The doctor or surgeon who prides himself on blunt speaking and scientific objectivity is a fool, and a cruel fool at that.

If the patient already knows he has cancer it should be explained to him that this word covers many kinds of disease some of which

are best treated surgically, and some by other means; and that in his case an operation will not be necessary.

In all instances, however, the closest relative concerned should be as fully and as accurately informed about the circumstances and the outlook as is possible.

Deep X-ray therapy

Irradiation is of considerable palliative value in the treatment of lung cancer but should not be used routinely before or after operations because so used it adds nothing to life expectancy and greatly increases the patient's discomfort.

A full course of deep X-ray therapy produces about the same physical disturbance as a pneumonectomy, and is therefore not in any way a milder or a more conservative substitute for surgery. No matter how skilfully high-voltage rays are administered they inevitably damage healthy lung irradiated at the same time as the tumour, and so add to a patient's breathlessness. This effect is worse in lower lobe lesions, where the rays have to penetrate more pulmonary tissue, than in upper lobe growths.

Squamous-celled carcinomas are only slightly sensitive to irradiation, and adenocarcinomas wholly insensitive, but undifferentiated oat-celled growths respond readily, though there is no evidence that its use improves their prognosis.

It is absolutely contra-indicated in the following circumstances:

- (a) If the growth has already disseminated.
- (b) If the growth is an adenocarcinoma
- (c) If sepsis is present in the lung
- (d) If the patient is already short of breath because of emphysema.

It brings relief, and is generally worth employing locally in restricted doses

(a) When there is pain due to inoperable chest wall invasion, with superior sulcus or vertebral involvement.

(b) When there is pain due to a metastasis in bone

(c) When there is mediastinal obstruction, especially if it is accompanied by pain.

(d) When there is difficulty in swallowing

Unfortunately ultimate recurrence of these symptoms is the rule after about three months, although by that time a cerebral metastasis, or death from some other cause, may end the patient's suffering.

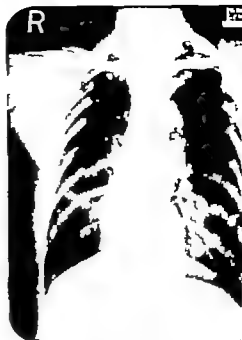
Irradiation is also of value:

- (e) in diminishing an exhausting cough;



57

57 A primary bronchial carcinoma in the lower lobe of the right lung. Now look at X-ray 58.



58

58. What has happened in the growth visible in X-ray 57? It has obstructed the bronchus to the right which has collapsed, drawing the heart across to the right, obliterating the cardio-phrenic angle and almost none.



59

59 A primary oat-cell carcinoma growing at the hilum of the left lung in a 19-year-old girl. W's emaciation is visible in both lungs. The disease presented with pain in the chest due to a pathological fracture of the 4th left rib.



60



61



62

1 A bronchial carcinoma of the right lung presenting by spontaneous pneumothorax. A mechanism seen in X-ray 36 has been set up but in this case the alveolar 'cyst' formed has ruptured into the p

2 Obstructive emphysema of right lung in woman with carcinoma in right main bronchus. On exph diaphragm rises normally, but right cannot because of air trapped in lung. Mediastinum is displaced to the left. On inspiration no abnormality was visible. Growth was removed by bronchotomy



63



64

Patient was man of 37 with inoperable oat-celled carcinoma obstructing left main bronchus. Whole l collapsed and mediastinum displaced to left. Note deviation of trachea and the fact that no heart is visible in the midline. Growth was treated by DXT. Left lung re-expanded, and pneumonectomy was performed (see X-ray 64)

See X-ray 63. The left lung has completely re-expanded and no abnormal opacity is visible in it. Mediastinum has returned to its normal position. A left pneumonectomy was successfully carried out

(f) in reducing unusually brisk or frequent haemoptyses.

It should be generally used in younger people who have un-celled growths, the size of the tumour being always greatly reduced. In addition they support irradiation better and it is either employed before surgery (when it may so shrink an inoperable neoplasm that it becomes removable later) or postoperatively to the mediastinum. When part of the lung is collapsed re-expansion can generally be obtained after deep X ray therapy (X ray 63) but when infection is present in it the treatment will have to be preceded by a course of antibiotics.

The nausea that frequently accompanies irradiation is mitigated by ensuring a daily fluid intake of not less than 2½ litres and by giving 60 mg. of pyridoxine three times a day.

Other methods of palliation

Intractable chest wall pain can sometimes be completely relieved by intercostal neurectomy (see p 75) (X-ray 65). But when growth erodes the vertebral column itself or involves the brachial plexus as it does in the Pancoast syndrome cordotomy may be necessary and well worth while.

Recurrent pleural effusions cause mediastinal displacement and breathlessness, and require repeated aspiration to keep the patient comfortable. Effusions of this sort are occasionally checked or diminished by the administration of radioactive gold.

Infection in the lungs or in the pleura causes much malaise, fever and purulent expectoration and a patient's condition can be greatly improved by control of this with antibiotics. Empyemas should be aspirated rather than drained, but sometimes insertion of a drainage tube is necessary especially in the presence of a bronchopleural fistula. The end of the drain must then be covered by an impervious dressing to allow the patient to breathe comfortably and to phonate.

Difficulty in swallowing which cannot be relieved by irradiation sometimes responds to dilatation of the stricture (which is usually caused by extramural pressure) with a Plummer bougie over a previously swallowed string. Gastrostomy is not really a palliative operation at all.

Improvement may also follow a four-day course of *nitrogen mustard*, 0.1 mg. per kilo of body weight being given intravenously each day. These courses can be repeated and I have seen considerable benefit result from them, albeit somewhat unpredictably.

Cortisone, in doses of about 25 mg. daily, is a valuable drug in terminal cases, the patients showing subjective improvement, gaining weight, and having less pain.

Other drugs which should be freely employed towards the end, as well as codeine and phenacetin (often as effective as any) are morphia and its derivatives, pethidine, methadone; heroin, or the Brompton cocktail, made of morphine, cocaine and gin. The doses of all these things should be as much as is necessary to achieve an effect. Finally, deprive no one of hope: it is the most valuable drug of all.

THE SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

THE introduction in recent years of effective anti tuberculous drugs such as streptomycin para aminosalicylic acid, isoniazid and others, has transformed the treatment and prognosis of pulmonary tuberculosis and represents one of the great medical advances of history. Before their advent 70 per cent. of patients who had a persistent tuberculous cavity in the lung died within five years. Medical treatment depended wholly upon 'rest', and surgery merely extended this policy in war extends the policies of diplomacy being confined to a variety of expedients aimed at resting the diseased lung and encouraging cavity closure, some of them minor in nature, some major, but all carrying potential dangers of their own and in any case quite inapplicable to the great majority of patients. At best none could be said to offer final cure, as in all the foci of disease were left behind to heal by natural processes and it was often difficult or impossible to be sure when this had occurred. As a result phrenic paralysis or artificial pneumothorax, although in theory temporary measures, were frequently in practice permanent and in minor cases liable to cause a degree of disability wholly out of proportion to the ill they were designed to cure. Worse still, even the most promising patients were necessarily confined to bed for very long periods, often for years, just at that period of young adult life when, apart from the evident economic consequences both to them and to the State, active life seems more precious, and mere survival a poor bargain. No other disease strikes as cruelly at the best age of man, and techniques which shorten the time needed for cure are of great worth.

A few years ago demand for beds far outstripped supply and many patients not only had to wait up to two years for admission to sanatoria but another two when they were inside for an operation which, if it was not a last resort, was often an inconclusive incident in a monotony of medical treatment. Today no sanatorium or surgeon need keep tuberculous patients waiting. Some hospitals have given empty beds to the rising flood of lung cancers and some lesser ones closed altogether. In addition there is a growing tendency now that hospital stay has been shortened, and sputum-conversion is more readily achieved, to treat more tuberculosis in general hospitals.

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rather than to segregate patients in the more remote centres or introverted little communities on mountain tops.

Pulmonary tuberculosis remains a difficult and formidable disease the treatment of which still calls for much time and patience as well as skill, and for which it is impossible to lay down therapeutic dogmas, as one can in connection with cancer of the lung, simply because the pattern of disease in each patient is individual and must be judged and treated individually. Whereas surgery has become vastly more important, more applicable and safe, and more conclusive in its results, it is certainly no simpler to apply. Tuberculosis is initially often widespread in the lung; even after it appears radiologically to be confined to one small segment, one cavity, one tuberculoma, the whole forest fire may be rekindled by an ill-judged or premature surgical attack. Less than 60 per cent of macroscopic lesions present are radiographically visible; and even after a perfectly planned and perfectly executed operation disaster may ensue if the remaining lung is not regarded as potentially infected, guarded against stress, and nursed gradually back to normal function.

Pulmonary tuberculosis not only affects the lung parenchyma but the bronchial tree as well, and it is certain that every patient has, or has had at some time, endobronchial disease. If this is confined to small distal bronchioles it in no way contra-indicates resection, but if ulceration is present near the site of intended amputation it does. Such matters must be decided by bronchoscopy beforehand; although it is just as disastrous to deprive a patient of appropriate surgery without good cause as it is to operate dangerously, and the mere fact that a mucosa which has been bathed in pus for many months is reddened does not necessarily mean that it is actively diseased and that operation must be postponed.

Every patient must still be prepared for a long period of bed rest. During the earlier and more acute stages of the disease this is necessary for the control of the outbreak, aided perhaps by antibiotics. Slow regression follows, and eventual localization to one or more segments of the lung where it may heal, or smoulder on to become a surgical problem. The more advanced and chronic patient, too, whose lung is already cavitated, must rest so that the activity of the disease and the size of the cavity, if one is present, are reduced to a minimum before surgery is contemplated. How long these processes take depends upon the patient's natural resistance and temperament, the extent of the lesion, and the virulence of the infection. They may be aided by posture, by specific drugs, or by minor surgical procedures such as the induction of artificial pneumothorax or pneumopcri-

toneum but major surgery ought never to be considered until they are achieved. After the most straightforward and successful major operation a patient must reckon on at least three more months in bed in complicated cases much longer.

Chest operations for tuberculosis are sharply divided into two groups—(1) those designed to relax the diseased lung so that it may heal by natural processes and (2) those in which localized disease is removed by pulmonary resection. Into the first group fall artificial pneumothorax, phrenic paralysis extrapleural pneumothorax, extrapariosteal plombage, and thoracoplasty, into the second pneumonectomy lobectomy segmental resection, and local excision.

Before the antibiotic era only relaxant operations were practicable, attempts at excision invariably resulting either in bronchial fistula formation or in the dissemination of disease. Ninety per cent. of all the major operations performed were thoracoplasties, the application of which as we shall see, is limited. It is clearly more attractive to extirpate diseased tissue than leave it behind in the hope that it will heal and today resections account for 90 per cent. of major tuberculosis surgery and thoracoplasty except as a purely secondary postoperative measure is an almost outmoded operation. Nevertheless, it must be remembered that *all* the disease present in the lungs is not removed by a resection that extirpative surgery has risks and troubles of its own, and that in certain cases a relaxant operation may still prove wisest and best. All pulmonary resections are performed under full antibiotic cover which has to be begun well before and continued long after, operation. It is therefore of truly vital importance that this weapon is not irreparably blunted in aimless medical treatment beforehand and organisms rendered resistant. When a tuberculosis patient enters hospital physician and surgeon must confer at an early date agree upon a comprehensive plan of action and carry it out together until the patient is discharged cured. It is a tragedy if the door of escape is slammed by lack of foresight and to become resistant to streptomycin is one of the worst misfortunes that can now befall a tuberculous patient.

One other important condition must prevail before any major operation either relaxant or extirpative, can be carried out there must either be no disease at all in the opposite lung or that which is present must be quiescent, or controlled by some lesser measure such as a mantle pneumothorax, an extrapleural pneumothorax, a small plombage, or limited apical thoracoplasty (X ray 79). To attempt a big operation upon one side in the face of active disease upon the other is to invite a dangerous flare-up of the latter with possibly

fatal consequences. It is true that this, like all rules in the art of medicine, can be broken occasionally with suitable help from antibiotics, and dramatic and immensely satisfying cures achieved by well-judged segmental resections first upon one, and then upon the other lung. In general, however, the rule should be observed.

RELAXANT OPERATIONS

These operations have always been strictly limited in scope, for they are of value only in closing chronic tuberculous cavities at or near the apex of the lung which have failed to close with more conservative treatment. They are of no value if the disease is solid, or if bronchiectasis or stricture is present, or when cavities are unfavourably placed, and of little value in the treatment of fibrocaseous lesions.

When a tuberculous lesion in the lung breaks down to form a cavity three things tend to make it bigger:

- 1 It may be increased by active tuberculous ulceration of its wall.
- 2 The lung is itself an elastic mesh in which the pressures ebb and flow from positive to negative during ordinary breathing. On inspiration the lung is sucked open like an accordion and is held to the expanding chest wall by the considerable negative pressure between the layers of the pleura. The effect of creating a hole in this balanced mesh is like making one in a gently stretched rubber sheet, a pin prick soon becoming a wide gap.
3. Small bronchi or bronchioles entering the cavity are compressed or partly stenosed by disease, so that air enters the cavity when they widen on inspiration but cannot escape again on expiration. We have seen this valve effect operate in other cavitating diseases and upon cysts of the lung. Cavities subjected to it increase in size as the tension within them increases with each inspiration, and even more with the deep intake of air that precedes a fit of coughing. The ballooning does not usually continue indefinitely because a state of equilibrium is reached when the positive pressure in the cavity is sufficient to force air out again (X-ray 72).

It will be seen therefore that the mechanical factor is very important, and as the lung is a three-dimensional elastic sponge it is imperative that its relaxation be concentric, that is, in all three dimensions and not only in two. This type of relaxation is well exemplified by a perfect pneumothorax without adhesions (X-ray 71); whereas a pneumothorax with apical adhesions may be much worse than none at all, for not only do the adhesions prevent relaxation in a vertical plane, but they probably run to the area most diseased and so in-

crease the stresses upon it (X ray 70) It is therefore useless to remove ribs and compress the underlying lung from side to side if the apical attachments of the pleural dome are not divided in order to free the lobe in its long axis also Again it is obvious that a pneumoperitoneum (which merely raises the diaphragm) is not likely to be so effective as a pneumothorax in controlling disease (X ray 74)

The induction and management of artificial pneumothorax and of pneumoperitoneum are minor procedures which are usually left to physicians. It should not be thought that because they are minor they are safe 40 per cent. of all tuberculous empyemas follow the induction of an ill-judged pneumothorax. Both are to be regarded as temporary measures, and two to three years the maximum period for their maintenance If the underlying disease has not healed by then they are unsuitable for its treatment and should be abandoned while it is still possible to do so A pneumothorax that becomes irreversible would have been much better never induced.

As relaxation must always be concentric if it is possible to make it so pleural adhesions found to be present when a pneumothorax is induced have to be cut, for whereas cavities close in about 60 per cent. of perfect A.P.s, they do so in only 25 per cent. of those with adhesions, and serious complications such as spontaneous pneumothorax, bleeding due to tearing of the lung, or empyema, are also much more likely in the presence of adhesions At this point the surgeon's aid is invoked. Adhesions are in fact present in the majority of pneumothoraces and are either visible directly on the X ray plate or their presence is indicated by impaired mobility of the lung on screening. Radiography however can never be relied upon as a measure of their true extent or of their divisibility and in all cases where they are suspected, or known to be present, thoracoscopy (which is a minor procedure) ought to be carried out. By it they may be cut, an unsatisfactory or dangerous pneumothorax converted into a perfect one and the patient possibly saved from a later major operation

Thoracoscopy and adhesion section

Providing a sufficient pleural air space exists into which to pass the thoracoscope there is no reason to delay more than a week or two in cutting adhesions after a pneumothorax is established Adhesions do not stretch and the sooner they are freed the better and the safer

The operation is performed under local anaesthesia, the patient lying on his side with his body arched over a sandbag or rubber rest,

and his uppermost arm extended to expose the axilla which has previously been shaved and surgically prepared with the rest of the chest. As adhesions are commonest about the apex, a high axillary intercostal space is usually the most convenient point for entry, but should they appear to be mostly elsewhere the space is chosen that offers best access to them, and it may be necessary to move from one space to another in order to reach new fields. The most satisfactory type of thoracoscope combines lighted telescope and electrocautery on a single carrier, so that the latter can be manipulated under direct vision.

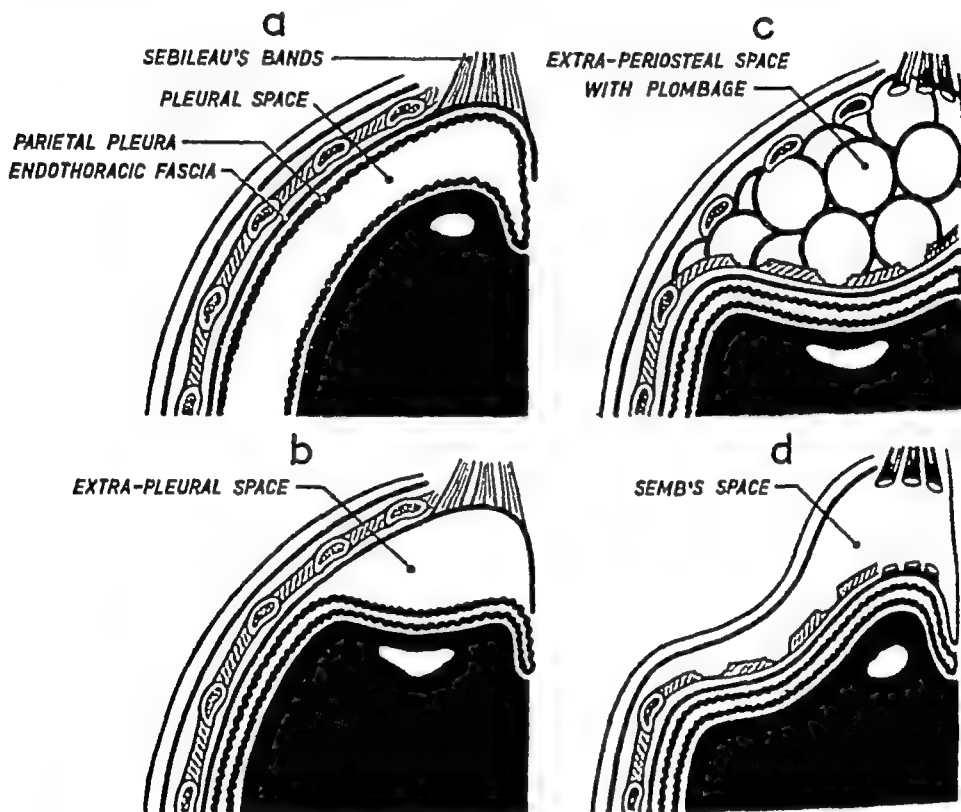


FIG 28

Diagrams to illustrate the anatomical differences between relaxant operations:

(a) *Artificial pneumothorax*: Air is admitted between the visceral and parietal pleural layers.

(b) *Extrapleural pneumothorax*: The parietal pleura is stripped from the endothoracic fascia to create an artificial space.

(c) *Extraperiosteal plombage*: The periosteum and intercostal muscles, as well as both pleural layers and the endothoracic fascia, are interposed between the diseased lung and the plastic balls.

(d) *Thoracoplasty*: The ribs themselves are removed. To relax the dome of the endothoracic fascia Sébilleau's bands (from the deep fascia of the neck) must be cut.



65



66

large carcinoma growing in the right lung. It has eroded the back ends of the ribs related to it. carcinoma growing at the very apex of the right lung and involving the upper part of the chest wall of growth which is certain to result in Pancoast syndrome. For its treatment see X-rays 67 and 68



67



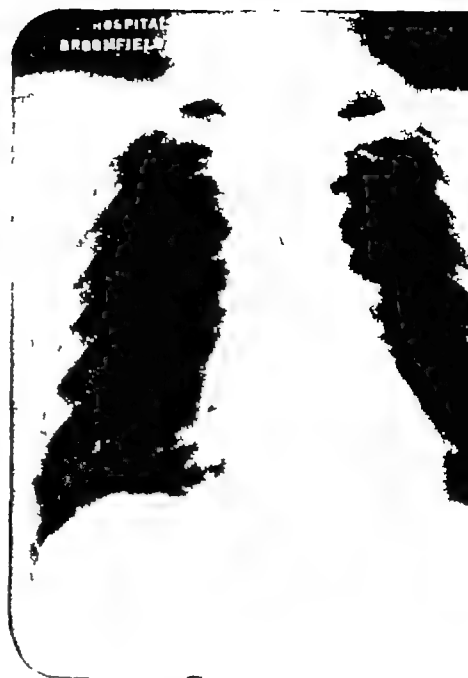
68

The growth seen in X-ray 66 has been treated by right pneumonectomy with resection of the upper chest wall including ribs, intercostal muscles and parietal pleura. For completion of the operation

after pneumonectomy and removal of the involved chest wall as seen in X-ray 67 the operation is right thoracoplasty to maintain the position of the mediastinum. The patient is alive and well



69



70

The next three X-rays dramatically show effects of relaxation in closing a lung cavity, and the divided pneumothorax. Here a large tuberculous cavity is visible in right upper lobe. It is suitable for treatment by pleurodesis, but resection is preferable. In fact a right artificial pneumothorax was induced (See X-ray 70). After induction of the right A.P. an adhesion is seen running to the wall of the cavity and pulling it into the shape of a pear. At any moment the cavity may burst and cause a tuberculous pyo-pneumothorax.



71



72

The adhesion seen in X-ray 70 has been cut in the nick of time. Now that concentric relaxation is achieved, the cavity has completely closed.

A giant tuberculous cavity at the apex of the right lung. Its size is largely due to a positive pressure within it caused by partial stenosis in the bronchus leading to it. This is well shown by what happens when a catheter-valve is inserted—in this case a Monaldi drain (see X-ray 73). Nowadays such a cavity is an indication for resection.



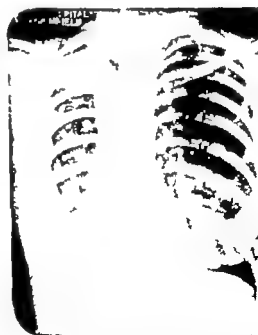
73. The cavity seen in X-ray N 72 after insertion of Mondri drain. As soon as the positive pressure is applied the cavity shrinks greatly in size. Primary excision is more effective still, for the bronchus communicates this cavity as partially stenosed.



74. A pneumoperitoneum in which the right phrenic nerve has been crushed in order to obtain greater elevation of the right diaphragmatic dome.



75. The appearance of the chest after right thoracoplasty. The right scapula lies against the mediastinum. Disease is present in the opposite lung. The right lower lobe is well aerated. The back is straight.



76. Healing of tuberculosis at the right apex by calcification and fibrosis. Note how the trachea is shifted to the right and how emphysematous the right lower lobe has become. A right apical thoracoplasty was performed.



77

77 Bilateral extrapleural pneumothorax has been carried out to control disease at both apices



78

78 An extraperiosteal plastic plombage has been performed to relax the apex of the left lung



79

79 Segmental resection has been performed upon the upper lobe of the right lung, lesser disease being controlled by an extraperiosteal plastic plombage



80

80 A destroyed left lung with a tuberculous empyema. A bronchopleural fistula is present for a long time. The contents of the empyema and its contents are being expectorated. Left pneumonectomy and total pleurectomy followed by left lateral thoracoplasty to preserve the right lung in which there is a very little disease

After the skin and parietal pleura in the selected space have been infiltrated with enough local anaesthetic to render the whole procedure completely painless, a short needle containing a light blunt probe is passed into the pleural space so that the distance of the lung from the parietes is ascertained. The skin over the anaesthetic bleb is nicked with a scalpel point, and a large trocar and cannula steadily thrust into the pleural cavity. The trocar is withdrawn, replaced by the thoracoscope, and the interior inspected. The condition of the pleura and the surface of the lung are noted, any free fluid present is aspirated through the sucker incorporated in the instrument, and if adhesions are present it is at once decided whether they can safely be cut. If this is not the case because the lung is directly adherent to the chest wall or mediastinum or from any other cause, the instrument is withdrawn together with its cannula, the puncture hole is sutured, a firm dressing applied, and the patient returned to bed.

If the adhesions appear safely divisible they are severed by the cautery (which is used at a red heat), as close to the chest wall as possible, or if they are very short, enucleated from it by cutting out a plaque of parietal pleura, to lessen the risk of burning the lung. The blood vessels they contain are never very big, but occasionally bleed briskly, and require coagulation. Bleeding is most likely if the cautery used is too hot, and the adhesions cut too fast or allowed to tear. As the lung relaxes, further sheets or cords of adhesions become accessible and the patient is placed in whatever position best exposes them, or the instrument is moved if need be to another intercostal space. It is for example helpful to elevate the table head so that the lung falls away from the apex and exposes the mediastinum. Success in cutting adhesions and in finding one's way round the pleural space by telescope is a matter of practice, and the experienced operator besides learning many personal tricks, is able to determine with accuracy what can safely and boldly be cut and what cannot. Generally speaking, if the lung cannot be wholly freed it is better left alone. After successful division the raw areas are inspected for oozing which may need coagulation. The instrument is withdrawn, intra pleural pressures adjusted and the entry hole closed with a stitch and a firm dressing.

Postoperatively the posture of the patient is supervised to prevent the lung becoming adherent to the operation site. For example, if the adhesions were chiefly in the right axilla, he lies on his left side or if on the right mediastinum, then propped up and inclined to the right to keep the lung as far as possible out of contact with the raw

surface. For the same reason it is important that the pleural space is well maintained, and this has to be carefully watched as there is a natural tendency for air to be coughed out of it into the tissues through the puncture wound. To avoid this the dressing is kept firmly strapped for the first few days and the patient is told to press his hand over it if he cannot suppress a cough. Coughing is kept at a minimum by the use of linctus and sedation, but as vomiting has much the same effect as coughing, opiates should be used with discretion. When air is coughed into the tissues it causes surgical emphysema, in mild cases a tender puffiness at the base of the neck giving characteristic crepitus on light touch. In more severe instances the face or even the whole body becomes swollen with subcutaneous air. Evidence of surgical emphysema is an indication for a pleural refill lest the space be lost, as well as for measures to prevent its recurrence. Air in the tissues is very uncomfortable for the patient, but providing the leakage does not continue, it is soon re-absorbed.

Air must be withdrawn from the space if postoperative radiographs show the lung to be collapsed against the mediastinum. It is very rarely necessary to bronchoscope patients after adhesion section, but if prompt re-expansion does not occur it may be necessary.

During the first twenty-four hours the pulse rate is carefully watched, for haemorrhage is the most serious and most common postoperative complication. It may occur from a vessel in one of the divided adhesions, but should not do so if they have been carefully coagulated and inspected at the end of the operation. Less easy to prevent is occasional oozing from the trocar wound itself. If blood accumulates in the chest it is aspirated completely and, if at all considerable, the loss replaced by transfusion. Any sign of continued oozing or of serious haemorrhage is an indication to return the patient at once to the theatre, reinsert the thoracoscope and cautery, and coagulate the vessels concerned.

Phrenic crush

The diaphragm is the most mobile and therefore the most important part of the chest wall from a physiological point of view. In the past, when other means of surgery were not available, or when satisfactory artificial pneumothorax could not be obtained, physicians frequently had recourse to phrenic paralysis, and even sometimes phrenic evulsion, in order to achieve some measure of pulmonary relaxation or to reinforce the effect of a pneumoperitoneum (X-ray 74).

The nerve was approached through a horizontal inch-long incision

three fingers breadth above the clavicle in the triangle made by the external jugular vein and the posterior border of sternomastoid. Platysma, fat and fascia were divided, or cleared by blunt dissection, and held out of the way by small lighted retractors. The surface of scalenus anterior then came into view with the nerve coursing medially upon it, passing deep to the transverse cervical artery which runs across the muscle at a right angle. The course of the nerve distinguished it from the cords of the brachial plexus emerging on the lateral aspect of the muscle and all passing downwards and outwards. The nerve was lifted up with a hook and crushed between the jaws of a Spencer Wells forceps.

The ensuing phrenic paralysis was intended to be a temporary measure lasting some three or four months but unfortunately it frequently proved permanent, and as it is quite impossible to guarantee recovery, this constitutes a very serious objection at any time to the operation, and an overwhelming one today when there is every likelihood of the patient ultimately needing major surgery. A paralysed diaphragm on the same side as a thoracoplasty or a pulmonary resection leads almost inevitably to basal collapse, as the patient is not able to cough up his secretions and this is doubly serious in tuberculosis for atelectatic lobes are very apt to become infected with tubercle bacilli. Ineffective coughing is also likely to result in spill over of tuberculous pus into the opposite lung with consequent spread of this disease. At best, the patient is rendered breathless and the maintenance of his heart and mediastinum in a central position after resection is difficult.

It is clear therefore that phrenic paralysis so easily carried out as a minor operation under local anaesthesia, may in fact gravely prejudice a patient's surgical future. The advantages gained by it are very uncertain and nowadays safer and more effective therapeutic methods exist. I do not think that under modern circumstances it is ever justifiable deliberately to paralyse one half of the diaphragm.

Thoracoplasty

Indications

The operation of thoracoplasty which used to be the chief weapon of the tuberculosis surgeon and accounted for 90 per cent. of his work, is now much less often used, having been almost wholly superseded by pulmonary resection, and to a lesser degree, by extra-periosteal plombage. The latter however is not always a completely satisfactory alternative as the degree of relaxation achieved by it is

not quite so great. For patients with bacilli resistant to antibiotics, and therefore unsuitable for resections, or in whom an apical and peripheral cavity exists which offers a particularly favourable opportunity for relaxant therapy, thoracoplasty is still of value. As it can be very gently staged, and even if it does not heal, increases the stability and 'safety' of underlying disease, it is useful also in some 'bad-risk' patients who would more safely sustain resection after preliminary thoracoplasty. It is also necessary to remove a varying number of ribs after many primary resections in order to obliterate dead space and so prevent overstretching and emphysema of the lobes left behind, or to promote the sealing of troublesome air-leaks. Sometimes this is done at the same time as the resection, but often it is left to a later date when the patient is better able to tolerate it and it is possible to see how well the remaining lung has adapted itself to the pleural cavity. Although it is of particular importance to prevent overstretching of the lung in tuberculosis lest latent foci are rekindled, emphysema must be avoided after any resection, and similar 'lateral' or modified thoracoplasties (i.e. ones in which only ribs are removed) are often necessary after lobectomies or pneumonectomies for non-tuberculous conditions.

A formal thoracoplasty involves not merely the removal of a number of ribs, but also complete mobilization of the apex of the lung, or *apicolysis*. This is essential to achieve concentric relaxation of the diseased area.

The operation is designed to close chronic tuberculous cavities of moderate size in the upper and outer third of the lung field—that is, in the apical or axillary portions of the upper lobe—which cannot be closed by other means. The rest of both lungs must either be free of disease or such as is present must be of very limited extent and under control (X-ray 69). Clearly it is undesirable to interfere radically with a growing skeleton, so thoracoplasty is quite unsuitable for young people, and it tends to be both disappointing and subsequently painful in the old. It is therefore most applicable between the ages of 20 and 40. Intelligent co-operation from the patient is of great value so he should both understand what is expected of him and be of good morale. He should also be gaining, or at least maintaining weight, and his erythrocyte sedimentation rate should be declining, or already low. Such are the ideal indications for thoracoplasty, but it is often necessary to depart from them.

The standard operation requires the removal of varying lengths of the upper seven ribs (X-ray 75). If fewer ribs than seven be taken the scapula is prevented from sinking medially against the mediastinum.

and insufficient relaxation of the upper lobe is obtained. Removal of the first five ribs is sometimes enough in dealing with very apical lesions or those in which a largely mechanical problem is presented, such as kinking of the trachea with traction upon the opposite lung by quiescent fibro-calcious disease (X ray 76), the scapula then remaining in its normal position.

Preoperative treatment

Removal of many ribs at once is incompatible with life, for the soft chest wall resulting moves in the opposite direction to the normal sucking inwards on inspiration and blowing outwards on expiration. This paradoxical movement, as well as greatly impeding normal breathing, makes effective coughing impossible, and secretions collect in the lung ultimately drowning the patient if he does not die first of respiratory failure. It is therefore essential to stage a seven rib thoracoplasty usually in two or three operations at intervals of a fortnight, so that the paradoxically moving part of the chest has time to become more rigid before additional ribs are removed.

This must be explained to the patient, and in the days preceding operation he practises diaphragmatic breathing and the maintenance of correct posture so important postoperatively if deformity is not to result. He is told also that after his operation the most essential single thing for him to do is to cough up his sputum.

Either general or local anaesthesia may be used but the latter has many advantages: shock and blood loss are less because of the adrenalin added to the infiltration solution; as the patient is conscious though drowsy his cough reflex is not seriously interfered with, and should tuberculous pus from the newly relaxed cavity spill into the bronchial tree he is able to cough it up without difficulty and so avoid the serious danger of aspirating it into his other and dependent lung; he is able to co-operate with the surgeon during the operation and at its conclusion can cough freely while still on the table and as soon as he gets back to bed, thus not only guarding against spill-over but also avoiding atelectasis of the lower lobe on the operated side. Collapse of this lobe is the commonest post-operative complication and calls for immediate bronchoscopy when it occurs.

A streptomycin screen is not usually needed in the type of case suitable for thoracoplasty but pre and postoperative penicillin cover is desirable and nutrition should be as good as the circumstances permit, the patient receiving 600 mg. of ascorbic acid a day.

to aid tissue repair. During the first stage a blood transfusion of at least a pint is given; quite often no more blood is required for the later stages

Premedication is heavy if local anaesthesia is to be used, and consists (for an average man) of Omnopon gr. $\frac{1}{2}$, with hyoscine gr $\frac{1}{160}$ given $1\frac{1}{2}$ hours before operation, and a further $\frac{1}{2}$ gr. Omnopon, three-quarters of an hour before. It should be in any case sufficient to ensure that the patient remains drowsy but just responsive.

Technique—first stage

The patient lies in the lateral position upon a chest rest on the operating table, the uppermost arm hanging freely forward, the hips held securely by special padded rests

250 cc of 0.3 per cent Xylocaine solution, to which 1 cc of fresh 1/1000 adrenaline is added, is used to infiltrate the skin and muscles along the line of incision, and to produce a paravertebral block of the first to the sixth intercostal nerves and of the vagus.

To secure good exposure of the first rib it is important to start the incision high, just below the upper border of the trapezius muscle, and above the superior angle of the scapula. The incision passes down midway between the scapula and the vertebral spines to curve round the inferior angle and run for a short distance along the line of the sixth rib

Trapezius and rhomboids are divided in the line of the incision, as well as a few fibres of the latissimus dorsi muscle; and the scapula is retracted forwards to expose the upper part of the thoracic cage. The wide origin of serratus anterior from the second rib is cut from it with a diathermy knife, and the fatty contents of the axilla, which became visible, swept forward by the finger so that the front ends of the upper three ribs are clearly seen. The periosteum over them is incised longitudinally with the diathermy, and stripped free with a rongeur from the costo-transverse joints behind to the cartilage in front. The second and third costo-transverse ligaments are cut with a chisel, the related rib necks with a rongeur, and both ribs are lifted forwards so that the soft tissue can be cleared from their under-surfaces and their costal cartilages divided. The whole of the first rib is now well displayed. Its periosteum is similarly stripped, first from the upper surface, with its scalenus medius attachments and rhomboid ligament insertion, then from the lower, and finally medially where the scalenus anterior muscle is inserted into its tubercle between subclavian artery and vein, both of which are intimately related to the rib. The costo-transverse ligament and rib neck

are dealt with as before, and the rib seized with strong forceps and disarticulated from its cartilage in front.

The whole of the upper three ribs have now been resected subperiosteally and a lateral, but not a concentric, thoracoplasty achieved. To complete the operation apicolysis is essential.

The first four intercostal bundles are divided as they emerge between the rib necks, and a plane of dissection is developed outside the *endothoracic fascia* which lines the parietal pleura. This fascia forms a dome over the thoracic outlet suspended by interdigitations of the deep fascia of the neck which pass down between the subclavian artery and the first thoracic nerve (closely related to the neck of the first rib as it runs up to join the brachial plexus) and in the angle formed by this artery and its internal mammary branch. These bands (sometimes called Sébileau's bands) are cut with scissors to let the whole fascial dome with both layers of pleura and the lung, drop freely down into the thorax (Fig. 28*d*).

This part of the dissection always requires care and is difficult if there is much fibrosis and induration of the tissues for it involves laying the following structures quite bare, in order from behind forwards the sympathetic chain, the first dorsal nerve trunk the subclavian artery giving off its internal mammary branch, which courses right across the mediastinum to run down the back of the sternum the vagus and recurrent laryngeal nerves and the superior vena cava or innominate vein, with the phrenic nerve lying on its surface. A little lower down the mediastinum, as it is exposed, one sees lying just in front of the vertebral bodies the oesophagus and the side of the trachea. The dissection is carried down the mediastinum until the arch of the aorta is encountered on the left side or the azygos vein upon the right—that is, virtually to the hilum of the lung (Fig. 29). The apicolysis is then complete and the whole upper part of the lung is lying concentrically relaxed in the thorax, covered on its apical and medial aspects by both layers of pleura and the *endothoracic fascia*, with, laterally the intercostal muscles and periosteum. An artificial cavity (called Somb's space) is left above and fills up with effusion which prevents the lung re-ascending (Fig. 28*d*).

The external muscle layers and skin are closed and the first stage is complete. A substantial pad of wool is placed over the now soft anterior and axillary part of the chest and firmly strapped up with Elastoplast bandages to control paradoxical movement. During the first twenty four hours this soft area must also be supported manually each time the patient coughs, as he is regularly encouraged to do. After the first day or so he learns to do this for himself.

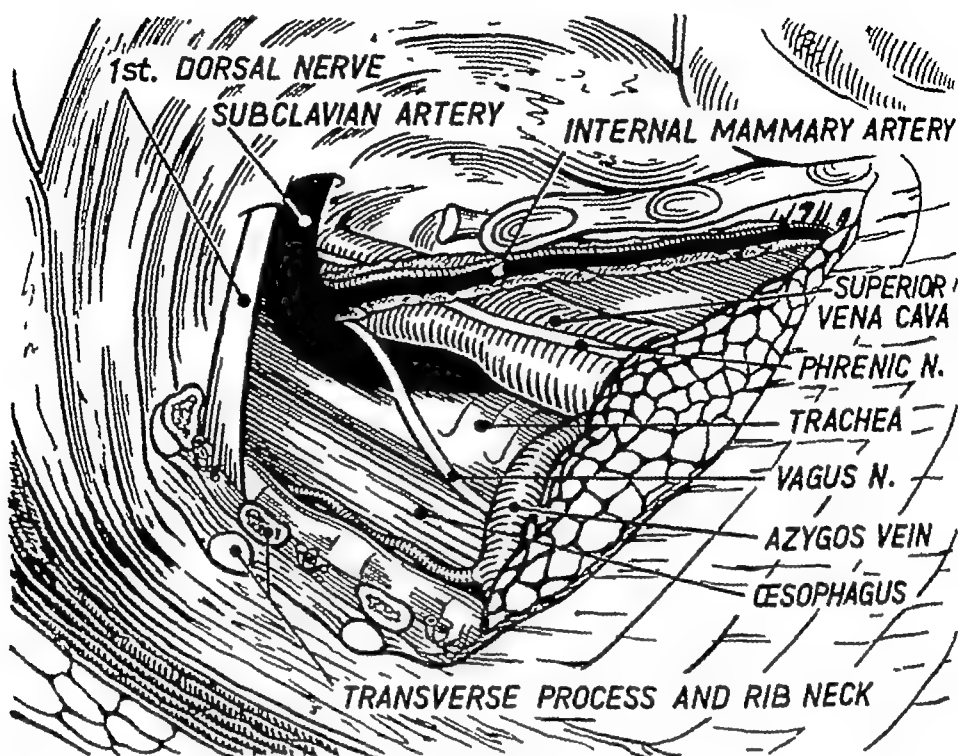


FIG 29

After first stage right thoracoplasty with apicolysis the upper part of the mediastinum lies bare. Behind is seen the first dorsal nerve ascending to join the brachial plexus, the subclavian artery, with the recurrent laryngeal nerve hooking round it, and giving off its internal mammary branch which runs forward and down the back of the sternum. On the superior vena cava lies the phrenic nerve.

Postoperative complications

(1) *Breathlessness*, or distress with *tachycardia* in the immediate postoperative phase, are most likely to be due to insufficient control of the paradoxical area; but other causes may have to be sought.

(2) If the parietal pleura is accidentally opened during the operation so that a *pneumothorax* is produced, the pleural air must all be removed with an A.P. apparatus at the end of the operation, but it sometimes happens that a tear passes undetected. If a portable X-ray subsequently reveals air in the pleural space it is removed.

(3) *Spontaneous pneumothorax*, due to a bleb rupturing either in the same or the opposite lung because of added respiratory strain during operation, or the effort of forced coughing after it, may cause very serious and abrupt dyspnoea. The accident is not common, but is most likely to occur in 'bad-risk' subjects, in whom it may prove

fatal if it is not promptly diagnosed and treated. This is especially likely if the intrapleural pressure becomes positive. An A.P. induction needle is inserted and all air removed, if necessary by continuous suction.

(4) Blood loss during operation is replaced as it occurs and pulse and blood pressure readings are taken at regular intervals afterwards. Serious *postoperative haemorrhage* is rare, but bleeding into the *Semb's* space sometimes occurs and considerable quantities of blood may collect in it. If bleeding is brisk it is accompanied by the usual signs of haemorrhage, requires replacement, and possibly the return of the patient to the theatre but if it is slow and insidious oozing, these signs may be absent, and breathlessness due to compression of the lung by the accumulated clot and to secondary anaemia predominates. The soft anterior chest wall bulges and relief follows aspiration of the blood through it, and suitable transfusion.

(5) Much the most common complication is *atelectasis of the lower lobe* on the side of operation. This is more likely to occur if the patient cannot cough up his sputum. If the diaphragm is paralysed collapse is almost inevitable. Diminution of air entry or impairment at the pulmonary base calls for confirmatory X rays and prompt aspiration by bronchoscopy if coughing fails to clear the lower lobe bronchi within a very short time. Collapse of this kind may be recurrent and calls for constant vigilance. Not all patients are breathless when collapse occurs, but most have a rise of pulse rate and temperature. Mediastinal shift may be slight. The essential points to be watched for are diminished air entry dullness and opacity of the lobe to X rays. Unless the lobe can clearly be seen to be aerated, *bronchoscope rather than tempotize*.

(6) *Spill-over*, or *spread* of the disease to the lower lobe, or opposite lung, or *reactivation* of disease already existing there. The first is seen early and is accompanied by fever and malaise the second later. Either is of grave significance and suggests that the selection or management of the patient has been faulty. Antibiotics should be started at once and all further surgery suspended.

(7) *Secondary infection of the Semb space* by pyogens, usually staphylococci, calls for increased antibiotic therapy drainage and obliteration of the space by completion of the thoracoplasty and embedding of the scapula. Tuberculous infection may follow the accidental opening of a lung cavity or even the rupture of contaminated lymphatics. Fortunately rare, it is an intractable disaster which may not respond to prolonged treatment with antituberculous drugs

Whereas pyogenic infections manifest themselves by fever, or by breakdown of the wound during the first fortnight, tuberculous ones are much more insidious, and are ushered in by unexplained low-grade pyrexia and malaise. Indolent discharging sinuses eventually appear in the line of the wound and communicate with the space

(8) *Nerve palsies* should never occur; but

(a) Horner's syndrome sometimes follows trauma to the sympathetic chain after difficult dissection of an indurated and fibrotic apex. It is usually transient

(b) The first thoracic contribution to the brachial plexus is vulnerable, and for that reason must always be particularly guarded

(c) The phrenic nerve is apt to become caught up in inflammatory tissue and to lift away from the mediastinum during dissection, when it may be injured. Providing this is known the accident will not happen

(d) Almost the worst injury that a thoracoplasty patient can suffer is damage to the long thoracic nerve of Bell supplying the serratus anterior muscle, paralysis of which results in 'winging' of the scapula and a most disabling loss of power in the arm affected. The loss of muscle substance also allows the scapula to rub painfully against the vertebral column. Although it is possible to relieve this pain by interposing a fat or muscle graft, 'winging' is virtually incurable

(9) *Chronic discomfort* after thoracoplasty is not as common as one might expect, but it is more likely in older subjects who already have arthritic changes. Wasting of the shoulder girdle muscles due to disuse promotes pain and can be corrected, and pain due to overriding of the angle of the scapula on an intact lower rib calls either for further rib resection or removal of the angle

Aftercare

In the very great majority of thoracoplasties the postoperative course is quite uncomplicated, except perhaps for transient collapse of the lower lobe. It is notable that this occurs inversely with the skill of the nursing, and in some units is quite exceptional. On the day after operation the patient extends his arms fully above his head and begins to move his shoulder freely. He has already been taught to overcorrect the posture to his head and neck *towards* the side of his operation, and so to diminish the tendency to scoliosis (with sagging of the head to the opposite side), which is the chief source of deform-

ity after thoracoplasty. A long mirror placed at the foot of the bed helps greatly in this. If joint mobility and good posture are made a constant care, disability or deformity need never be feared and when they are seen they stand as monuments to the surgeon's neglect.

At the end of a fortnight the patient, with straight back, full arm movement, good aeration of the lower lobe, paradoxical chest movement much diminished, and the wound healed, should be ready for the next stage. If a period longer than two or at the most, three weeks elapses, the planes of dissection are lost, and there may even be new bone formation from the displaced periosteum of the resected ribs. Providing the first operation has caused little upset and the patient is fit, it may be decided that the thoracoplasty can be completed at once but if he is older short of breath, and the lung moved very freely during apicolysis it is safer (and on the whole more usual) to perform three stages. In elderly emphysematous or bronchitic patients it may be necessary to stage the operation at an even gentler tempo, removing perhaps only two ribs at the first stage, and so on.

Technique—second and third stages

At the second stage the wound is re-opened, the accumulated serum and clot cleaned out of Serratus space, and the apex of the lung re-mobilized if it has risen in the thorax and become re-adherent. As a rule there is little or no bleeding, as vessels have not yet crossed the lightly healed planes and further transfusion is seldom needed.

The posterior three-quarters of the fourth and fifth ribs are resected as before, and the chest reclosed. If it is desired to embed the scapula at once and complete the thoracoplasty, the back halves of ribs 6 and 7 are similarly treated otherwise they are dealt with in a third and final stage a fortnight later.

A 7 rib thoracoplasty of this kind diminishes a patient's vital capacity by about a third. This is well tolerated by most people in their late twenties and thirties but progressively less well after middle life. Formerly thoracoplasties involving 8, 9 or even 10 ribs were commonly performed. This should never be done today. If a 7 rib collapse seems inadequate to deal with the disease thoracoplasty is not the right operation for such a case, and resection is indicated instead.

Results

If a thoracoplasty fails to close the cavity for which it was performed the sputum remains positive and this constitutes the test of

success: should the sputum prove positive on rigorous and repeated *culture* three to four months after operation, the thoracoplasty has failed, and resection of the underlying diseased lobe or segments is indicated without further delay.

In properly selected cases a competently performed thoracoplasty of the type described can be relied upon to produce permanent and consistent sputum conversion in more than 80 per cent of subjects. The complication rate is very low indeed, and the mortality rate is in the vicinity of 2 per cent.

Lateral thoracoplasty

Following lung resections, both tuberculous and non-tuberculous, it is often desirable to obliterate the dead pleural space to prevent overstretching and emphysema of the residual lobes or lung, and to keep the heart and mediastinum central. To achieve this, ribs are removed in the manner described above, their number varying with the space to be filled. Quite often the scapula can remain in its normal position. It is also an advantage to leave the first rib in place, as an apicolysis is not required, and the attachments of the scalenes to it diminish the tendency to deformity. The pleura over the thoracic outlet can be peeled down without disturbing the rib (X-ray 38).

Revision thoracoplasty

In the past when a thoracoplasty failed to close an underlying cavity a 'revision' was often undertaken to improve the degree of relaxation. The previous wound was re-opened, and the irregularly re-formed ribs either excised, or the whole acquired bony carapace overlying the lung apex removed *en masse*. The operation no longer has a part to play. Its results were always most uncertain and today an unclosed cavity is an indication for resection.

Extrapleural pneumothorax

This operation has now fallen almost wholly into desuetude and in any case has enjoyed a bad reputation for many years in England, partly because it was often applied to unsuitable cases, partly because of its dangers and the difficulties of its aftercare. In spite of this, however, and of its very limited applications, it has a unique advantage: it is reversible (see Fig. 28b).

Indications

The type of lesion for which it may be employed is the small sub-apical chronic cavity, not too peripherally placed, and with minimal

surrounding fibrosis which cannot be treated by artificial pneumothorax. If such a lesion exists with more serious disease in the opposite lung demanding resection, or the small cavity is mirrored by an almost exactly similar contra lateral one, extrapleural pneumothorax may provide a solution to the difficulty. For on the one hand it is necessary to bring disease in one lung under control before embarking on major surgery on the other and bilateral segmental resections or apical thoracoplasties may be undesirable methods of dealing with small equal lesions, although bilateral extrapleural operations which can be given up after a year or two when the disease has healed are acceptable (X ray 77)

Technique

The back ends of the upper five ribs are exposed under local anaesthesia, in the same way as for a thoracoplasty but through a much shorter incision. A few inches of the third or fourth ribs are resected subperiosteally and the deep layer of the periosteum and the endothoracic fascia incised. The plane between the fascia and the parietal pleura covering it is then developed by blunt dissection and the parietal pleura stripped off the chest wall and thoracic outlet with the finger thus creating an artificial space. When this is big enough, a single rib spreader is inserted to give better access, and the dissection is carried over the apex and down the mediastinum until the upper part of the lung is relaxed downwards, covered by its two adherent pleural layers. With the aid of a malleable light and a dissecting swab on forceps, the strip is completed until the lung is mobilized down to the level of the fifth rib. Bleeding points are coagulated as they are met with, and occasional strands of fibrous tissue have to be cut with scissors.

Aftercare

It will be seen that such an operation is quite unsuitable for anyone with gross or long-standing disease and a hard fibrotic apex or for those in whom active tuberculosis is so near the surface of the lobe that it might contaminate the space. The tendency of a dead space such as this to become secondarily infected (either by tubercle or pyogenic organisms) constitutes one of the chief objections to the operation. Another is the risk of haemorrhage, for even after meticulous haemostasis, reactionary bleeding from the chest wall into the space sometimes occurs, a large clot collecting which may strip down the rest of the pleura, promoting fresh bleeding as it does so and compressing the affected lung to cause severe dyspnoea. An event

of this sort demands prompt transfusion, evacuation of the clot, and control of the bleeding points.

In uncomplicated cases serum collects in the space and must be aspirated postoperatively and replaced by air. These aspirations are continued until the space is quite dry and well established, after which periodic refills are given with an A P apparatus to a positive pressure of about 20 or 25 cm of water, this being the only time that air is admitted to the chest under a positive pressure. Refills are carefully maintained, probably for several years, until the lesion is judged to be healed and the sputum is consistently negative; or until major surgery on the opposite lung has been successfully concluded. They are then stopped, the air in the space absorbs, and much of the collapsed apex re-expands.

Extraperiosteal plastic plombage

This operation combines some of the features, and avoids some of the disadvantages, both of thoracoplasty and of extrapleural pneumothorax, and is tending to replace them both. The degree of relaxation it achieves is not quite so complete and thorough as that gained by a good thoracoplasty; and it differs in one important respect from the extrapleural operation—it is not reversible.

To eliminate the necessity for air-refills attempts were often made in the past to fill extrapleural spaces with vegetable oil ('oleothorax') or paraffin wax ('wax plombage') (X-ray 29); but the usual result in both was either secondary infection, or ulceration of the foreign substance into the lung and its subsequent piecemeal expectoration. With the advent of non-irritating plastics these were employed instead. As at first many suffered a similar fate, attempts were made to increase the safety of the operation by interposing more tissue between the diseased lung and the prosthesis. In addition to the parietal pleura, the endothoracic fascia, the intercostal muscles, and the periosteum covering the upper five ribs were all stripped down on top of the lung to form a solid barrier, and the plombage was inserted between them and the bare ribs outside (Fig. 28c). The use of the periosteum means that new bone forms on the lung side making its collapse permanent. Various types of plastics were tried, polystyrene sponge (which has the disadvantage that should infection occur it is difficult to remove); polythene bags stuffed with polythene ribbon to produce the right degree of tension; and lucite balls pierced by cylindrical holes to help accommodate any accompanying effusion. These balls have the great advantage of being easy to insert, and can be increased or decreased so as to fill the space snugly and

maintain just the required volume without pressing unduly upon the lung (X rays 78, 79)

Indications

The indications for extraperiosteal plombage are similar to those for thoracoplasty although as a rule it is prescribed for less severe and less long-standing cases in whom radical relaxation of the upper lobe is not required. It is often carried out to control apical disease on one side of the chest preparatory to resection upon the other, but the relaxation obtained is permanent. It has the advantages over thoracoplasty that it can be carried out in one operation, that no ribs are removed, and therefore there is no risk of deformity, and as there is no soft chest wall and the plombage prevents free excursion of the lungs, paradoxical movement is eliminated.

Technique

The surgical approach is the same as that for extrapleural pneumothorax and local anaesthesia is used as before. No rib resection is necessary. The periosteum is incised over the upper five ribs and is stripped only from the under-surface of the first rib, but from the whole of the remaining four. The intercostal bundles are secured and divided near the rib-necks so that an unbroken but mobile sheet of intercostal muscles and periosteum clothes the outer aspect of the lung. The apex is freed extrafascially and when a sufficient degree of relaxation has been achieved an intercostal space is spread, and the lucite balls are inserted through it to lie between the bare ribs externally and the musculo-periosteal sheet internally the latter together with the endothoracic fascia and both layers of pleura, providing a barrier between the prosthesis and the lung. When enough balls have been added just to fill the space, the ribs between which they have been passed are allowed to fall together the external muscle layers and skin are closed and the operation is complete.

Aftercare

Complications after extraperiosteal plombage are fewer and less serious than after either thoracoplasty or extrapleural pneumothorax, though this is partly because of the type of case chosen. Excessive effusion into the space may cause pressure symptoms, and require aspiration and secondary infection is not unknown. In the latter event the balls or plastic prosthesis must be removed, the space drained, and later obliterated as far as possible by thoracoplasty.

The troublesome bleeding and pleural stripping that may accompany extrapleural operations is not met with.

RESECTION

Relaxant operations are valueless for lesions situated elsewhere in the lung than in the upper and outer portions of the upper lobes; and even then they are suitable only for the closure of cavities, although they promote healing in fibro-caseous disease to some extent. The ability to remove diseased tissue altogether, irrespective of where it may be or of its particular character, enormously extends the range and usefulness of tuberculosis surgery. There are, however, certain limitations and risks which do not apply, or do not apply so forcibly, to pulmonary resections for other conditions. For instance, tuberculosis is never really limited to one lobe or segment of the lung, and therefore prolonged antibiotic therapy and postoperative rest are still needed to ensure that it does not flare up elsewhere after the more obvious lesion has been excised. Similarly the parts of the lung left behind must be most carefully guarded against overstretching, so that it is often necessary to perform subsequent apical or lateral thoracoplasties to obliterate dead space. Finally, although the risk of postoperative bronchial fistula is small providing the organisms remain sensitive to antibiotics, it is still somewhat higher than that for non-tuberculous resections. In addition, persistent air-leaks from the parenchyma and very small bronchioles are a troublesome though not particularly dangerous feature of segmental resections, which nowadays make up the great majority of pulmonary excisions for tuberculosis. This is because, although a cavity, or tuberculoma, or whatever the lesion be, is confined to a given segment that is removed, there is usually some fibrosis or secondary emphysema or other pathological damage in neighbouring segments, which have been left raw.

It is always the thoracic surgeon's aim to remove the minimum quantity of lung tissue that ensures extirpation of significant disease. This is most important in tuberculosis patients, whose future is often uncertain and who may require bilateral resections. In all cases the remaining lung is able to accommodate itself much more easily to the loss of a segment than to that of a whole lobe. In a few patients, such as those in whom a whole lung is utterly destroyed and useless, pneumonectomy is demanded; in more, a complete lobe is involved and lobectomy required, but in 80 per cent. of cases it is sufficient to excise one or more isolated segments.



81 Following a right artificial pneumothorax the upper lobe proves unexpandable. It should be resected (X-ray 82.)



82 The same patient as in X-ray 81 after right upper lobectomy. The middle and lower lobes now fill the cavity.



83 A small tuberculoma is visible in the axillary part of the left upper lobe in the fifth interspace. The sputum is positive. Should anything be done about it? For the consequence of doing nothing turn to X-ray 84.

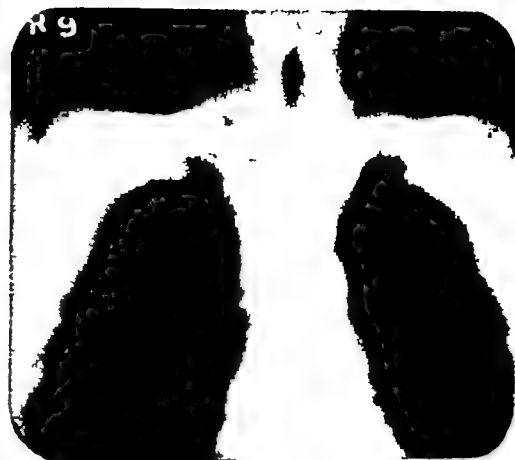


84 The tuberculoma seen in X-ray 83 has broken down into a tuberculous cavity and serious apical emphysema has occurred by aspiration from it.



85

X-ray 85 Tuberculous bronchostenosis causes collapse of this middle lobe. The patient presented with haemoptysis and the sputum was negative until after bronchoscopy, when the stenosis was slightly dilated and pus was able to escape. Middle lobectomy is indicated.



86

X-ray 86 Tomography of a tuberculoma at the right apex shows it to be cavitated, in spite of the fact that calcification is also present in it. It should be excised.



87

X-ray 87 A tuberculous empyema of long standing in a patient with negative sputum. Decortication of the chronically thickened pleura was carried out and the lung re-expanded as seen in X-ray 88.



88

X-ray 88 The lung seen in X-ray 87 after decortication.

Indications

The best time for operation whatever its scope, is chosen by physician and surgeon working in concert. Preferably the activity of the disease is at its minimum. The patient has long been afebrile, his sedimentation rate is steadily declining, or already low, his weight steady or rising. Tubercle bacilli however, are found in his sputum, or can be cultured from it, unless they have already been banished from it by streptomycin. To operate upon a patient whose sputum had *always* been negative, on the other hand, calls for very special consideration: are the dangers of leaving the lesion in his lung alone greater than the small, but definite, risks of the operation? Are the lesions of a kind which experience shows will probably cause trouble though they may be causing little now? What is the natural resistance of the patient like? Can he earn his living? Does she want to get married and have children? Have they any common sense?

Most patients submitted to the surgeon have already had considerable quantities of streptomycin and para-aminosalicylic acid or INAH, but if they have not, a preoperative course of streptomycin with one or other lasting not less than six weeks is desirable, streptomycin being given in doses of 1 G daily with 5 G of para-aminosalicylic acid six hourly or 200-300 mg. INAH daily. Should sensitivity tests show the patient to be resistant to streptomycin, recourse can be had to isoniazid (100 mg. b.d.) viomycin or Marnalid. Drug resistance is so grave a drawback that it makes most forms of resection impracticable.

The scope of pulmonary excision for tuberculosis is being constantly extended and still varies a good deal from surgeon to surgeon (see Fig. 30). About the following indications there is, however, no debate.

(1) *The so-called "destroyed" lung*. Quite often patients are seen in whom one lung is honeycombed with cavitation, or collapsed and clearly functionless, whereas no disease is evident in the opposite one or is confined to a few quiescent foci (X ray 80). The prognosis in such cases is desperate if nothing is done but pneumonectomy followed by lateral thoracoplasty to preserve the integrity of the remaining lung, frequently transforms the outlook. Even if such patients preoperatively are rather breathless, or have some bronchospasm in the good lung, removal of the diseased and functionless one by diverting the wasted blood flow through it, thereby improves oxygenation, and reduces breathlessness. Bronchospasm may prove to have been largely a reflex from the pathological side.

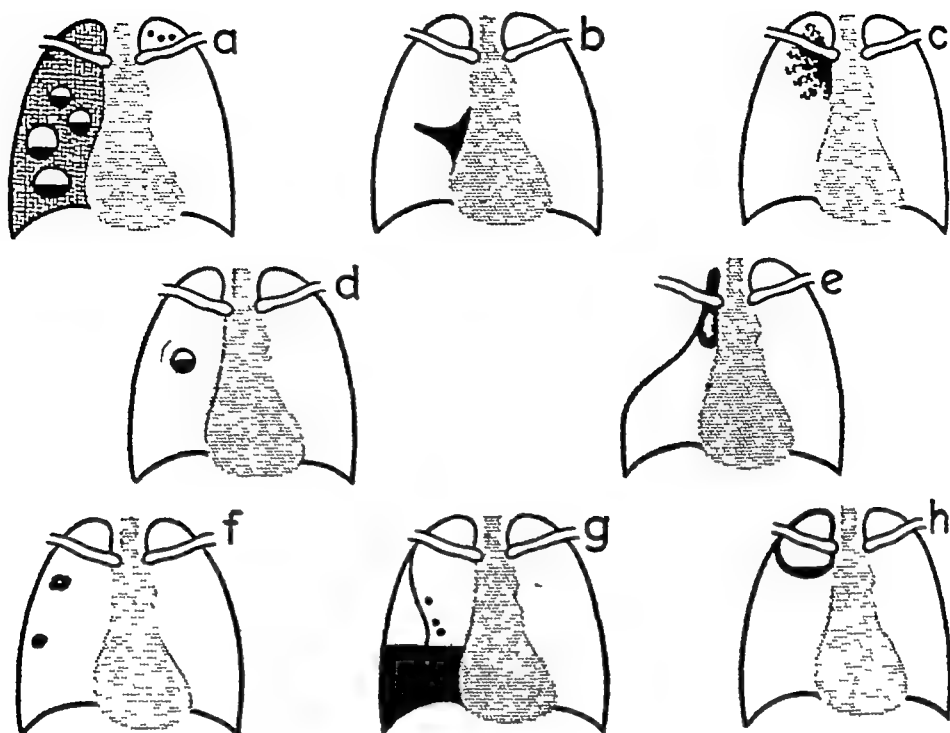


FIG 30

Some of the indications for resection in pulmonary tuberculosis.

- (a) A destroyed lung
- (b) Persistent collapse
- (c) Tuberculous bronchiectasis
- (d) A chronic cavity unsuitable for treatment by relaxation.
- (e) A failed thoracoplasty
- (f) A tuberculoma (cavitated here)
- (g) A tuberculous empyema with bronchopleural fistula.
- (h) A giant cavity

(2) *Persistent collapse of a lobe* or the presence of *tuberculous bronchostenosis* 'Black' lobes following artificial pneumothorax, or atelectatic lobes which will not re-expand after bronchoscopy and chemotherapy, are due either to extensive parenchymatous disease or to bronchostenosis (X-rays 81, 82, 85) If they cannot be relieved, the least of the troubles that overtake them will be bronchiectasis, and excision is the best treatment. Breathlessness is then less for the reasons already mentioned.

(3) *Tuberculous bronchiectasis*. In addition to patients like the above, many with long-standing disease have suffered complete or partial obstruction to bronchi at some time or other which has left them with a bronchiectatic lobe or segment, and they continue to produce tuberculous sputum from it although no obvious cavity is

present. Bronchography is of value in defining exactly the limits of the damaged area. Relaxation is clearly pointless in such cases.

(4) *Chronic tuberculous cavities* These may be of a nature or in a position unsuitable for thoracoplasty. Giant cavities which occupy virtually the whole of an upper lobe (X ray 72) or those with greatly thickened and indurated walls, or those situated close against the mediastinum, or in the apex of a lower lobe or in a basal segment, are all best treated by removing the affected segment or segments.

(5) *Failed thoracoplasty* This is one in which the sputum remains positive direct or on culture 3-4 months after the conclusion of the operation. When this proves to be the case no more of the patient's time should be wasted: the offending lobe or segment beneath the thoracoplasty is removed. Access to the chest is a little more difficult, but the actual resection is not, and the postoperative disturbance is usually considerably less.

(6) *Solid lesions or tuberculomas* No form of collapse therapy can possibly benefit the solid foci so commonly seen in the lungs of patients with pulmonary tuberculosis. Some of these lesions result from resolving exudative disease: others appear unheralded, especially in the lungs of young people, and fade capriciously without treatment and without apparent reason: and at least 10 per cent. of them are simply inspissated tuberculous cavities.

There are therefore many instances in which their removal is quite unjustifiable. Providing always that they are known certainly to be tuberculous, nodules of less than about 3 cm. in diameter should be left alone, as should those showing signs of regression or of calcification, or for which there is X ray evidence of stability for many years.

The circumstances under which they should unhesitatingly be excised (assuming them to be the sole, or at any rate the predominant, lesion present) are:

(a) If they are the source of *positive sputum* or *haemoptysis* (X rays 83-84). The fact that the sputum is negative is, however, by no means a contra-indication to their removal, especially if antibiotics have previously been given: for tubercle bacilli can be found in 80 per cent. of smears taken from the interior of removed tuberculomas in patients whose sputum was negative.

(b) If they show radiological evidence of *increase in size*, *alteration of edge* or if tomography reveals them to be *cavitated* (X ray 86).

(c) If being without signs of calcification, they are *larger than 3 cm.* or so in diameter, for experience shows the larger lesions to be

more likely to cause trouble. Even calcification is not an invariable sign of safety.

(d) *If any doubt exists about their identity.* This becomes of very serious importance if the patient is male and over 40, as the risks of any rounded opacity in the lung fields after this age being a peripheral carcinoma are considerable.

In a consecutive series of 50 patients of all age groups with such single opacities in their lungs, sent to me from mass radiography clinics, 38 per cent proved on removal to be carcinomas, and three of these latter patients had accompanying evidence of old tuberculosis, one actually having a positive sputum.

(7) *Bronchopleural fistula.* The presence of a *bronchopleural fistula* or a *tuberculous empyema associated with a positive sputum* in which the disease is confined to one lobe or lung. If a fistula exists the patient not only inevitably has an empyema, but coughs pus up from it, and a fluid level is radiographically visible (X-ray 80). Spontaneous pneumothorax resulting from rupture of a tuberculous cavity calls for prompt excision of the cavitated segment. An empyema may also simply be secondary to active tuberculosis in the subjacent lobe from which positive sputum is produced. In these cases, providing the condition of the rest of the lung permits, the best treatment is resection of the diseased area and removal of the empyema by total pleurectomy. When a tuberculous empyema exists, but sputum is *negative*, and disease in the underlying lung is healed, decortication is indicated instead, with removal of the empyema and re-expansion of the lung.

Preoperative treatment

Prior to operation, in addition to having antibiotic therapy, breathing exercises, and if necessary postural drainage and blood transfusion, every candidate for resection should be bronchoscoped to ensure that there is no active tuberculous endobronchitis at, or proximal to, the intended site of amputation. Recognition of this calls for judgement and experience, and many patients are rejected for surgery quite unnecessarily simply because their bronchial mucosa is somewhat reddened.

Technique

The technique of pulmonary resection for tuberculosis differs little from that already described for non-tuberculous conditions. Segmental resection is by far the commonest operation, and in the removal of lobes or whole lungs the approach to the hilum is made

both easier and safer by stripping the parietal pleura from the chest wall and reaching the bronchus and main vessels wholly in the extrapleural plane. The operation then becomes one of total pleurectomy with pneumonectomy or lobectomy. If the disease in the lung and pleura is very long-standing technical difficulties can be formidable. One consequence of extrapleural approach is an increase in the degree of shock and in considerable oozing from the chest wall. These call for extra vigilance in blood replacement both during and after operation.

In very exceptional circumstances it is permissible to perform a strictly local removal of a peripheral and apparently inactive tuberculoma, either simply by shelling it out of the parenchyma as one does a chondromatous hamartoma, or by excising the surrounding fringe of tissue with it by wedge resection between clamps.

Aftercare and complications

These are exactly the same as for non tuberculous resections with some added hazards.

(1) As in all operations for tuberculosis *spread or reactivation of disease* in other parts of the lungs may occur. If spread is due to the spill-over of tuberculous pus during the operation it is manifest in the dependent segments of the opposite lung in the first few post operative days but reactivation of previously latent disease may not be apparent until much later. Either calls for intensified antibiotic treatment and long continued rest.

(2) *Bronchopleural fistula* is now fortunately rare, but is still rather more often seen after resections for tuberculous than for non tuberculous conditions. It is most likely to occur in those with some degree of resistance to antibiotics, in the debilitated or exsanguinated, in those with undetected tuberculosis at the site of amputation, or those in whom amputation of the bronchus has not been flush, and a dependent stagnant pocket has been left. The fistula is sometimes much delayed, occurring weeks or even months after operation, but otherwise exactly resembles in its symptoms and consequences fistula formation as already described following non-tuberculous resection.

If the onset is promptly detected the best treatment is immediate thoracotomy with resuture or re-amputation of the leaking bronchus which should be covered by an intercostal muscle graft if this has not previously been done. It is also desirable to perform a lateral thoracoplasty to bring the chest wall into close apposition with the stump. Unfortunately such expedients do not always succeed, or a

tuberculous empyema is already established before anything can be done

(3) *Persistent bronchiolar air-leaks* These have already been mentioned as the most common and troublesome complication of segmental resections, but because they occur much more peripherally in the bronchial tree than do the type of bronchial fistula described above they are not infected, and consequently do not cause empyemas (see p. 200)

To provide some indication of the risks of resection for pulmonary tuberculosis I find that in 200 consecutive operations performed in the past three years five instances of bronchopleural fistula occurred, though all survived, and the fistula was successfully closed in three of them by resuturing and limited thoracoplasty. Of the remaining two, one developed an empyema which was subsequently drained. The space was much reduced by thoracoplasty, eventually sterilized, and the patient is now well and sputum-free. Three of the five patients were either wholly or partially resistant to streptomycin

Ten patients, most of them having had segmental resections, had troublesome postoperative air leaks of the bronchiolar type which called for further major surgical treatment, either reopening the chest and sewing up the leak, or apical thoracoplasty, or both

Seven deaths occurred within four months of the 200 operations, that is, a hospital mortality of about 3.5 per cent

Tuberculous empyema

Tuberculous empyemas are disasters whose occurrence is frequently preventable, and whose cure is sometimes impossible and always difficult. The principles governing their treatment are essentially the same as those applied to other types of empyema, but the special nature of the infection and the need for dealing also with the disease in the underlying lung impose certain differences in management

The need for timely prevention is indicated by the fact that so high a proportion of tuberculous empyemas complicate pulmonary disease already under treatment. For example, 40 per cent of them follow the induction of an artificial pneumothorax, generally one in which adhesions persist or in which attempts to divide them have failed—in short they are man-made. It follows that artificial pneumothorax must never be regarded as a form of minor therapy to be embarked upon lightheartedly, but calls for well-judged and skilful management, and unremitting supervision. At the first danger signs

of fluid accumulation in the pleural cavity or evidence that it is otherwise ineffective or unsatisfactory it ought promptly to be abandoned.

Should pus, whether it be sterile or not, collect in the pleural space it must at once be completely and, if necessary, repeatedly aspirated in the hope of securing re-expansion of the lung and its early adherence to the chest wall with consequent obliteration of the space. This is in exact accord with the treatment of acute non-tuberculous empyemas. Providing the case is a sufficiently early one, persistent efforts along these lines will succeed and constitute the ideal cure. Unfortunately pleural thickening and fibrosis occur rapidly and to an extreme degree in tuberculous infections and render both lung and chest wall immobile at an early stage so that the lung is rigidly corseted and cannot re-expand (X ray 87). Under these circumstances, which are the rule rather than the exception a drainage tube should never be inserted, for not only will it become a permanency but it will also probably lead to secondary infection of the space by other organisms. Should secondary pyogenic infection, however be superadded from some other cause a drain becomes essential until more definitive treatment can be undertaken, especially as patients with such empyemas are particularly likely to develop amyloid disease.

The circumstances under which pulmonary resection with total pleurectomy is indicated in the treatment of tuberculous empyema have already been mentioned. If active disease is present in the lung beneath the empyema, as evidenced by positive sputum, re-expansion of such a lung is contra indicated, and the procedure should be lobectomy or pneumonectomy (according to the location and extent of the active disease) coupled with pleurectomy i.e. removal of the empyema cavity *en masse* by stripping the parietal pleura enclosing it from the chest wall. Separation in this extrapleural plane is carried on round the chest, sometimes with great difficulty but, fortunately, as the pulmonary hilum is approached it is generally free of the intense fibrosis encountered elsewhere, and isolation and ligation of the vessels can readily be carried out.

For patients in whom the chronic empyema itself is the problem, and in whom the underlying pulmonary disease is healed and the sputum negative only the surgical removal of both layers of thickened pleura by decortication provides any hope of eradicating it and of restoring normal function. The performance of a thoracoplasty designed to obliterate an empyema cavity by bringing the chest wall down to the imprisoned lung, instead of expanding the

latter to the former, merely adds still more to the impairment of function without getting rid of the empyema

Decortication can be a formidable and is always a major undertaking. The removal of greatly thickened and very hard parietal pleura involves both shock and blood loss, and the patient's general condition and haemoglobin level must be as satisfactory as possible beforehand. Antibiotic cover is advisable.

A postero-lateral thoracotomy is performed, usually with removal of the rib most centrally situated over the empyema, although it is better if rib resection can be avoided altogether. The abscess cavity is at once opened widely, all the pus and fibrinous slough inside it are sucked out, and its limits defined. The parietal extrapleural plane is found and the whole tough mass stripped manually, or if necessary by sharp dissection, off the chest wall up to and including the angles of return on to the lung or the mediastinum. It is during this phase that bleeding may be severe, and adequate provision must be made for blood replacement. Hot towels are packed into the freed areas. If the diaphragm is involved, it too should be cleared, but the process is often more difficult over it, and it is important that its musculature is not damaged. On the mediastinal aspect the greatest care must be taken not to injure the phrenic nerve directly, by stretching, or by the use of diathermy coagulation near it.

When the parietes are clear of thickened pleura, the layer overlying the lung is deliberately incised down to the surface of the lung when a visceral plane of cleavage is reached. This is in turn developed by sharp dissection (which unavoidably results in some superficial alveolar air-leaks), or by peeling it off in the less closely attached parts with a swab. Where it is densely adherent over areas of old disease, however, it is best left undisturbed as an isolated plaque. In particular it is cleared out of the fissures and from the numerous folds in the collapsed lung surface. Eventually the whole fibrous carapace is removed and the lung is liberated.

The anaesthetist now applies positive pressure to re-inflate the lung; and the degree to which he succeeds in this depends upon the amount of healed disease and fibrosis within it (X-ray 88). If this is minimal, almost complete re-expansion is possible even though the lung has been collapsed for a long period. A dependent and an apical underwater-sealed drainage tube are inserted before the chest is closed, and both are connected with continuous suction which is maintained until X-rays show the lung to be everywhere in apposition with the chest wall. If residual air spaces become cut off it may be necessary to empty them by a separately inserted intercostal drainage

tube, for should either fluid or air be allowed to accumulate between the lung and the chest wall re-expansion is prevented, thickening and fibrosis recur, and the end result will be disappointing. Every effort is now made to obtain, and to preserve, mobility of the chest wall and of the diaphragm, and exercises for both are done hourly by the patient and twice a day under the instruction of a physiotherapist. Spreading of the narrowed intercostal spaces is obtained by the patient lying with his affected side arched over sandbags or pillows.

The aftercare of patients who have had a decortication is at least as important as the operation itself and all the advantages gained by the latter are forfeit if lung expansion chest mobility and correct posture are neglected.

TUMOURS AND CYSTS OF THE MEDIASTINUM

THE mediastinum contains an extraordinary variety of important structures: the heart itself; the great arteries and veins, the principal lymph channels and their rich effervescence of attendant nodes, segmental, sympathetic and parasympathetic nerves; air passages, the trachea and main bronchi, the oesophagus, glands of internal secretion, the thymus and often the thyroid and parathyroids. Any one of these diverse tissues may give rise to a pathological mediastinal mass.

In addition the mediastinum is an arena for complex developmental events. It is upon this narrow stage that the more caudal of the branchial arches unite; that the primitive enteron buds off its pulmonary diverticulum, which in turn branches and divides into the ultimate elaboration of the bronchial tree; that the foregut itself differentiates, that the simple tubular heart undergoes its foetal mutations, convolutes, and descends into the thorax carrying other structures with it; that the pleural and pericardial sacs are fashioned by infoldings of the simple coelom, and the whole cut off by the formation of the diaphragm from the septum transversum. Any one of these processes may give rise to mediastinal abnormalities, usually cysts but sometimes tumours.

Finally the mediastinum, from its central position, frequently becomes a repository for lesions originating elsewhere: for directly invading lung cancers; for secondary deposits from growths of the lungs, or of abdominal organs, or its rich lymphatic tissue is involved by generalized disorders such as the reticuloses, lymphadenoma, or tuberculosis, which may give rise to craggy masses due to adenitis, or to abscesses. Protrusions of stomach or of gut are often thrust into it from below, encysted effusions or empyemas in the pleura become intimately related to it; scoliosis or vertebral disease distort it; and even herniation of a lung across the midline may resemble a mediastinal cyst wall.

Not only, therefore, are mediastinal tumours and cysts extremely common and of great variety, but their differential diagnosis embraces virtually every endothoracic disorder; and provides a fascinating study, which calls in the first place for accurate history and careful and full physical examination.

Other investigations must always include good lateral, and, if

necessary oblique X rays as well as postero-anterior views. The position of tumours in the mediastinum provides the best guide to their identity (see Fig 31). Radiological screening is indispensable for testing mobility and watching pulsation. It may thus be possible to identify suspected aneurysms of the great vessels, or of the heart, and to demonstrate their continuity with vascular structures. The confirmatory evidence of serological reactions and characteristic physical signs are needed but it must be remembered that very

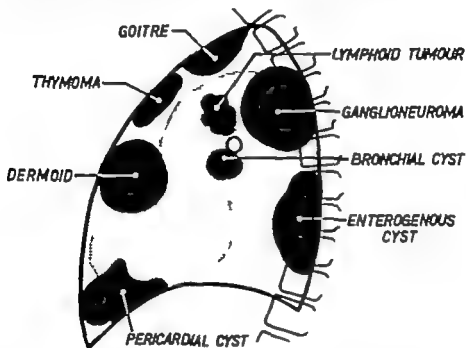


FIG 31

A diagram showing the characteristic position of common tumours in the mediastinum as seen in a lateral X-ray

vascular growths pulsate directly as well as by transmission, and that a loud bruit is often audible over them so that in the final resort angiography is the arbiter. Kymography also helps to define pulsation of a tumour and postero-anterior or lateral tomography determines the exact relationship to neighbouring structures or demonstrates cavitation within the mass.

Interpretation of X rays in infants can be very deceptive because it is difficult to obtain a strictly centred film: the mediastinum is highly mobile at this age and minor rotations produce gross alterations of outline which are easily mistaken for 'tumours'.

Barium swallows and meals are valuable in excluding oesophageal lesions and diaphragmatic hernias, in detecting indentation or pressure upon the oesophagus from without, and in testing the movement of related masses during deglutition. If long-standing cardiospasm is present the greatly dilated oesophagus takes up an S-shape, bulging to the right in its upper portion and to the left lower down, so that when full of food it much resembles a mediastinal tumour.

Bronchoscopy is always essential to exclude primary bronchial carcinoma; and may reveal pressure upon a bronchus from without. Oesophagoscopy plays a similar role. Occasionally the induction of an artificial pneumothorax enables one to distinguish between a tumour in the lung or in the mediastinum, although thoracoscopy is seldom of value. A pneumoperitoneum sometimes helps in defining the relationship of masses abutting on the diaphragm (X-ray 97).

After all tests are completed and extraneous growths and diseases have been eliminated, one is left to disentangle the wide variety of intrinsic mediastinal cysts and tumours. Very often a tumour is manifest merely as an abnormal radiological shadow, and it is possible to do no more than guess at its nature from its general character and its position in the mediastinum. Rarely can there be certainty of diagnosis and still less often assurance of innocence or malignancy. Of all mediastinal tumours at least 10 to 15 per cent. are cancerous or are likely to undergo malignant change. All are liable to enlarge, and cause breathlessness by their mere bulk in the pleural space, or by pressing upon the adjacent trachea or bronchi; dysphagia by compressing the oesophagus; engorgement of the neck veins and tachycardia by obstruction of the great vessels and displacement of the heart; and eventually all these by generalized mediastinal obstruction. Innocent neurogenic growths may result in compression of the spinal cord.

Cysts are prone to secondary infection, and are then apt to rupture into the pleural cavity or into the lung with disastrous consequences; or establish fistulous communications with the bronchi and behave thereafter like chronic abscesses. Intrathoracic goitres may cause obstruction or become secondarily toxic. Eighty per cent. of thymomas are associated with myasthenia gravis, and in any case should be considered malignant. Enterogenous cysts may undergo peptic ulceration and perforate. Hydatids may disseminate their infestation.

It is folly to treat such tumours with complacency, to await their growth, degeneration or infection; to stand idly by 'observing' them while something easily and safely removable turns into something formidable or fatal. As soon as they are found they should be

removed and if the patient has no symptoms at the time so much the better. Where is that skilled clinician who can tell with certainty an innocent dermoid from a malignant thymoma? He does not exist.

Common tumours of the *anterior mediastinum* are, from above down

- 1 Intrathoracic goitres.
2. Thymomas.
- 3 Dermoids and teratomas.
- 4 Pericardial coelomic cysts

They must be distinguished principally from bronchial carcinomas, with sarcomas of the sternum, aneurysms of the first part of the aorta, and hernias through the foramen of Morgagni a long way behind. Lipomas are rare, but are also found anteriorly related to the cardio-phrenic fold.

In the *posterior mediastinum* are found

- 1 Ganglioneuromas
2. Neuroblastomas (in infants)
- 3 Enterogenous cysts.

Here again carcinomas of the lung provide the chief diagnostic problems, together with encysted effusions or empyemas, aneurysms of the descending aorta, passive distension of the oesophagus, posterior congenital diaphragmatic hernias, and tumours or abscesses associated with disease of the vertebra.

In the *central mediastinum* occur

- 1 Most of the tumours of lymphoid tissue
2. Bronchogenic cysts.

The differential diagnosis in this situation is made particularly difficult by the frequency with which lung cancers and metastatic deposits in the glands occur.

The relative frequency of these cysts and tumours is not easily determined owing to wide variations in pathological diagnosis, and to the fact that their recognition is comparatively recent. Substernal goitres are commonest of all, but are not wholly mediastinal and are therefore omitted from the account.

Among 45 cases of my own 20 were neurogenic tumours, and of these 14 (31 per cent.) were ganglioneuromas 2 neuromas (of which one was sarcomatous) 2 manifestations of von Recklinghausen's disease, and 2 neuroblastomas 6 were thymomas 5 dermoids or

teratomas; 3 bronchogenic cysts, 3 pericardial coelomic cysts; 2 enterogenous cysts; 2 lymphadenomas, 1 lymphosarcoma; 1 lymphangioma, 1 haemangioma, and 1 true ectopic thyroid. Twenty-two per cent. were therefore malignant. Of 105 mediastinal tumours removed in United States Army Hospitals in three years 90 were innocent, 15 malignant. Among the innocent were 29 ganglioneuromas, 23 bronchogenic cysts, 14 dermoids and teratomas, and 10 pericardial cysts, the malignant included 6 teratomas, 4 lymphadenomas, 2 lymphoblastomas and 2 thymomas. 160 cysts and tumours from the Mayo Clinic records showed roughly similar proportions.

THE ANTERIOR MEDIASTINUM

Intrathoracic goitre

Ordinary colloid goitres do not have to increase very much in size before parts of them become retrosternal; but quite often the main bulk of the goitre lies wholly or partly behind the sternum in the anterior part of the superior mediastinum and is then easily mistaken for an intrinsic tumour. As the thoracic outlet at this point is limited and rigid, early symptoms of mediastinal obstruction such as distension of the neck veins and discomfort on bending down soon occur, perhaps to be followed by general breathlessness, dysphagia, and swelling of the face if the gland continues to increase in size. A sudden increase of this kind may follow haemorrhage into one of the colloid cysts, so that a patient hitherto almost symptomless is abruptly reduced to suffocation. The trachea is always displaced to one or other side according to the thyroid lobe chiefly enlarged, and if it is also narrowed, or flattened from in front, dyspnoea and stridor follow. When secondary toxic degeneration takes place in colloid goitres the chief effect is felt by the myocardium and atrial fibrillation results.

Most patients have some palpable evidence of a goitre in the neck, or a previous thyroidectomy scar, but in those with neither, the retrosternal mass can usually be felt to lift on swallowing, or on deep inspiration, against a finger placed in the suprasternal notch or the supraclavicular fossae. X-ray screening is always necessary and should be combined with a barium swallow to confirm that the shadow does rise on deglutition; failure to do so suggests either that the tumour has become malignant or that it is not in fact arising from the thyroid.

Radiologically, retrosternal goitres appear as lobulated shadows behind the upper part of the sternum in postero-anterior views,

almost always more prominent on one side while pushing the trachea to the other and having a clean edged convexity downwards (X rays 89 90) The upper pole however, shades off into the neck and cannot be defined. On the lateral view the opacity is seen to lie anteriorly and it is again its lower edge which can best be seen. Some long-standing goitres show irregular patchy calcification which makes them easy to identify (X ray 90) The use of radioactive iodine isotopes which are taken up by thyroid tissue is sometimes helpful confirmatory evidence in doubtful cases

Operation

The surgical approach to retrosternal goitres is always by means of a collar incision in the neck as for an ordinary thyroidectomy, as the vascular attachments of the gland remain in the usual place no matter how big the goitre gets or how deeply into the thorax it appears to thrust. Sternum splitting is very rarely required indeed and should be performed only as a last resort.

The distended superficial veins are tied or avoided and the infra hyoid muscles divided on whichever side is the chiefly enlarged lobe. The superior thyroid artery and vein are dissected out ligated and cut, and the inferior thyroid artery ligated in continuity as far away from the gland as possible in order to avoid damage to the recurrent laryngeal nerve as it courses up on the postero-lateral surface of the lobe. The inferior thyroid veins, which are usually stretched over the retrosternal mass, are secured and divided one by one and by a mixture of steady traction on the gland with the left hand and leverage from the right index finger which sweeps round the capsule behind the sternum, the goitre is delivered into the neck. In the great majority of cases this does not present serious difficulty but in exceptionally large adenomas it may be necessary to evacuate some of the contents of their cysts before bringing them up.

One or if necessary both, the lobes are then partially resected in the same way as for an ordinary subtotal thyroidectomy.

True ectopic thyroids that is an intrathoracic thyroid gland whether enlarged or not, without normal vascular attachments in the neck, are exceedingly rare and cannot be diagnosed prior to removal.

Thymomas and thymectomy

Myasthenia gravis is associated with disorder of the thymus gland. An active principle can be obtained from fresh glands by acetone extraction, one intravenous dose of which will paralyse experimental animals for periods of up to twenty minutes and which is also found

to make muscles much more sensitive to the effects of tubocurarine. Although the thymus is relatively larger in childhood, it by no means atrophies in later life, remaining a substantial bilobed structure weighing an average 15 G in the adult, and lying in the anterior mediastinum just behind the sternum and overlapped by layers of the pleura from both sides of the chest. At its upper pole two small cornua extend up into the base of the neck, approaching the thyroid isthmus; while the lower lobes lie upon the front of the pericardium. The left innominate vein passes immediately deep to the gland, receiving from it a short thick vein, and its arterial supply is by a number of very small twigs from the internal mammary vessels.

In 85 per cent of people with myasthenia gravis no physical abnormality is apparent in the thymus, yet after thymectomy nearly 70 per cent of such patients are able to live normal lives either with no neostigmine at all or with only occasional doses.

In the remaining 15 per cent. an epithelial tumour or 'thymoma' is present, and when this is so they respond less well to neostigmine before operation and recover less satisfactorily afterwards, and the disease itself is usually particularly severe, abrupt in onset, and more likely to prove fatal. Surgical removal, preceded by deep X-ray therapy, however, still secures comparably good results in about 55 per cent. of cases. The difference in prognosis of the two groups is significant.

In addition to those thymomas associated with myasthenia gravis, others occur, histologically identical, in which there are no symptoms of the disease, but the growth is discovered either on routine radiography, the onset of substernal pain, or because of early mediastinal obstruction resembling that caused by a retrosternal goitre. If such tumours are not removed, some patients later develop myasthenia, but some die of direct extension or metastases without any evidence of it. The true incidence of these 'silent' thymomas is difficult to determine, for many have been classified as malignant lymphoid tumours in the past, but it seems probable that some three-quarters of them all are associated with myasthenia, so that any abnormal anterior mediastinal shadow in a myasthenic patient is virtually certain to be a thymoma, and retrosternal pain in such a patient is always of grave significance.

Quite apart from their connection with myasthenia gravis all thymomas should be regarded as malignant tumours and therefore demand urgent removal. They are slow growing, composed of epithelial cells and thymocytes palisaded round cystic spaces and blood vessels. Hassall's corpuscles are usually wanting. The whole is



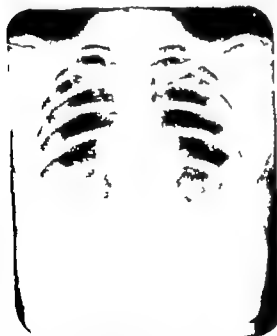
89

X-ray 89. A front view of a typical retrosternal goitre. Its upper pole cannot be distinguished. It is displaced to the left.



90

X-ray 90. A retrosternal goitre in the anterior superior mediastinum. Irregular calcification is present. Diagnosis.



91

X-ray 91. A thymoma associated with mild myasthenia.



92

X-ray 92. A malignant thymoma in characteristic position in the anterior mediastinum. Nonetheless distinguishable from an innocent dermoid at this stage, for the patient had no myasthenic symptoms.



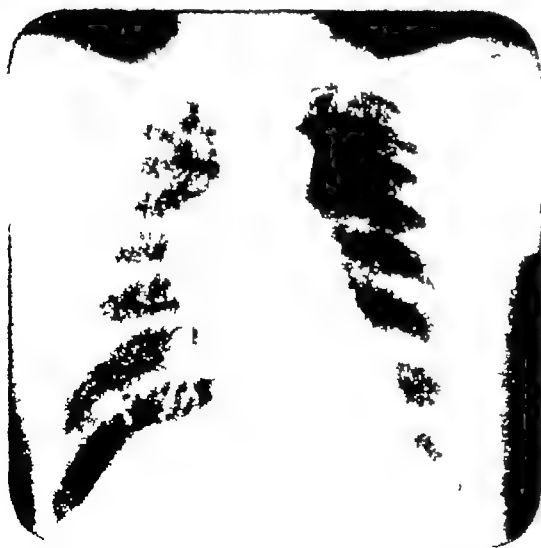
93



94

X-ray 93 An anterior view of the calcified dermoid cyst seen also in X-ray 94 Although the cyst lies in the mediastinum it bulges more to the left than the right side in this instance

X-ray 94 An oblique view of a dermoid cyst in characteristic position in the anterior mediastinum Note how the heart and great vessels are displaced backwards The wall of the cyst is calcified making its character certain (See also X-ray 93)



95



96

X-ray 95 A dermoid cyst of the mediastinum bulging this time to the right

X-ray 96 A pericardial coelomic cyst lying in the anterior part of the cardio-phrenic angle A pneumothorax has been induced to demonstrate its relationship to the diaphragm (See also X-ray 97)



ray 97. A lateral view of the same cyst seen in X-ray 96. Note its anterior position to the base of the mediastinum.
 ray 98. An anterior view of a typical ganglioneuroma. Its nature only becomes apparent when a lateral view is obtained. (See X-ray 99.)



ray 99. A lateral view of a typical ganglioneuroma. Note its position in the paravertebral gutter and its smooth edge. Compare with X-rays 98 and 100.
 ray 100. A enormous ganglioneuroma half-filling the left pleural cavity. Note its characteristic posterior position in the paravertebral gutter and its smooth edge.



101

X ray 101. A neuroblastoma in an infant's left thorax. This tumour lies in the paravertebral gutter like a neuroma. It is speckled with fine calcification—in spite of its being highly malignant. The child presents paraplegia due to an intrathecal extension of the growth. There is smooth pressure erosion of the related



102

X ray 102. An enterogenous cyst containing gastric mucosa on the right side of the posterior mediastinum infant and closely attached to the oesophagus. Note the accompanying deformity of the thoracic cage



103

X ray 103. A large enterogenous cyst causing lymphangiomatosis



104

X ray 104. A large enterogenous cyst in an adult. It contained thick mucus and was closely attached to the oesophagus.

wrapped in a thick fibrous capsule and so may appear innocent, but later they erupt through this invade adjacent structures such as the pericardium directly and some finally metastasize. Even calcification which is occasionally seen in them, is no guarantee of innocence.

It is imperative to treat all thymomas with high voltage irradiation before undertaking their removal and it is therefore essential to determine whether or not one is present in every myasthenic patient before performing a thymectomy. Failure to do this greatly worsens the prognosis, but should it happen that a gland is removed and subsequently discovered to contain a growth or a mediastinal tumour found to be a thymoma, the area must be irradiated afterwards.

On ordinary postero-anterior X ray films the tumour is obscured by the sternal shadow unless it is very large when it appears as a lobulated mass on one or both sides of the sternal edge (X ray 91). Providing focus and penetration are correct, the great majority should be detected by lateral views, and are often characteristically flattened against the back of the sternum in the upper mediastinum (X ray 92). Tomography provides confirmatory evidence. In contrast to retrosternal goitres the convexity of the upper pole of the tumour is visible, and they neither rise on deglutition nor are palpable in the neck.

When myasthenic symptoms are present the diagnosis is not in doubt and deep X ray therapy is begun at once to be followed on completion and irrespective of the response by thymectomy. If there is no myasthenia the tumour must equally be irradiated and removed together with the gland, not only preventively but because it is malignant.

Thymectomy

Immediately prior to operation the patient, if myasthenic, is given an injection of neostigmine equivalent to his oral dose. The use of curare in the anaesthetic is to be avoided, induction being by Pentothal and followed by gas and oxygen. The patient lies on his back with the head slightly raised, and the skin and subcutaneous tissues in the suprasternal notch and down the midline of the sternum are infiltrated with local anaesthetic. A vertical midline incision is made from one inch above the manubrium to the level of the fourth costal cartilages. This is deepened to incise the periosteum of the sternum which is then stripped off on either side for about an inch but cleared right out to the edge of the sternum at the third intercostal space. A blunt dissector is passed across the back of the sternum from the

right third space to the left, keeping close to the bone; and the tissues similarly cleared from the suprasternal notch until a finger-tip can be passed down behind the sternum. The blunt foot of a Schumacher's sternum splitter is passed into the notch and down the back of the bone which is cut in the midline as far down as the level of the third spaces which are then joined by a transverse cut. Bleeding from the bone edge can be controlled by Horsley's bone wax. A retractor then separates the two leaves of the sternum and lays bare the anterior mediastinum (see Fig 32). Right and left pleural folds almost meet in the midline and must be gently swept to either side with a dissecting swab. Should either be torn the hole must be at once stitched up and any air in the pleural cavity subsequently removed. The thymus is identified by its two pink, fleshy lobes and cleared of areolar tissue and fat. Its lower pole is lifted and dissected off the pericardium until the two veins running on the under-surface of its lobes are seen to join into one short vessel which drains directly into the innominate vein passing obliquely across the thymic bed (Fig 32). This thymic vein is doubly ligated and cut, and finally the whole gland, together with any tumour it may contain, removed, only a few small arterial branches requiring ligation at the periphery. After the mediastinum has been inspected for haemostasis and for any pleural leak, the retractor is removed, and the two leaves of the sternum are approximated, and held in position by sewing together the two layers of the periosteum. It is not necessary to fix the halves by bone sutures, which in fact contribute to postoperative pain. The superficial tissues are sutured and the skin closed with Michel clips. If drainage is employed the tube is led out of the lower end of the wound and underwater-sealed lest any air-leak has been overlooked. The dressing is kept in place by a St Andrew's cross of strapping so that it does not interfere with respiration. A further neostigmine injection equalling the usual oral dose is given, and oral administration resumed when consciousness is regained. Postoperative neostigmine requirements sometimes rise sharply in a so-called 'myasthenic crisis'. When a large tumour is present the procedure is similar, except that it may be necessary to split the sternum farther down, and the subsequent dissection of the mass may be much more difficult and involve the removal of neighbouring structures.

The postoperative care of myasthenic patients is almost wholly concentrated on preserving their lungs from collapse by keeping their airways clear, and by promoting effective coughing. Many of them already have difficulty in coughing because of their muscular weakness, and they have in addition a tendency to excessive secretion of

mucus. Any factor which makes their position still worse such as air or effusion in one or other pleural cavity, may therefore prove

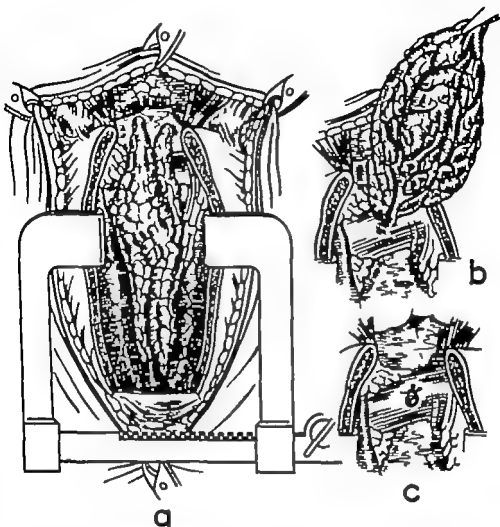


FIG 32

The operation of thymectomy

(a) The sternum split and retracted to show the bilobed thymus gland lying in the superior mediastinum overlapped by both pleurae.

(b) The thymus has been dissected from the pericardium and turned back to display the thymic vein (of Keynes) entering the innominate vein.

(c) The vein has been ligated and cut and the thymus removed.

fatal and this is the reason why every care must be taken during thymectomy not to damage the pleura, or should it be damaged, to repair it and subsequently to remove air or fluid by aspiration. Post operatively neostigmine requirements are constantly watched and

palpitations due to cardiac displacement, breathlessness or substernal pain. Once this period is over they may remain silent, to be discovered only on routine radiography, or until such time as they are overtaken by one of the complications to which they are prone.

Of these infection is common in the case of dermoids and occurs readily without any prior fistula, being presumably blood-borne. In the first instance this leads to malaise and a low-grade fever of unknown origin, together with a formidable increase of the adhesions binding the tumour to its surrounding structures. If the infection is severe it can result in rupture of the pus into a bronchus, or the pleural space, or the pericardium, with disastrous results. Sometimes bronchial fistulae develop without evident infection so that the contents of the dermoid are expectorated bit by bit, and sebaceous material and hair appear in the sputum (trichoptysis), thus leading in dramatic fashion to the diagnosis. Hairs may also be aspirated directly from the cyst, or from the pleural cavity after rupture. Occasionally dermoids present in the base of the neck, or beside the sternum, and either spontaneously, or after puncture, discharge their characteristic contents upon the surface. Once a sinus or fistula is established secondary infection is inevitable and the cyst behaves thereafter as a chronic abscess.

A blonde young woman of 23 took a fall out hunting and was thought to have cracked a rib. Her chest was X-rayed and most of the left pleural cavity found opaque. Haemothorax was then diagnosed and an attempt at aspiration made. No blood was obtained, but two dark hairs were impacted in the withdrawn needle. At operation a huge shaggy dermoid half filled the left chest and appeared densely adherent to a much thickened pericardium. This later proved to be another dermoid to which the first was attached by a stalk, like a dumb-bell, the pericardium being grossly displaced to the right.

Apart from such dramatic evidence as the aspiration or spitting up of hairs, diagnosis depends chiefly on radiographic appearances. These consist of a rounded or lobulated shadow, always anterior in the mediastinum, but bulging to one or other side of it, and in its earlier phases having a clearly defined edge. Later on successive attacks of inflammation reduce this sharpness so that the edge becomes almost as blurred as that of a carcinoma. Once a fistula is established there is a fluid level of mucus or pus; but even before this happens the fatty contents of the cyst may separate out at body temperature and float on the more aqueous material to produce an apparent level called 'Phemister's line'. Teeth are occasionally visible in der-

moids or, more commonly a regular line of punched-out translucences due to tooth buds in the midst of a dense germinal ridge

If the wall calcifies it establishes the shadow as a cyst (X rays 93-94) but must be distinguished from calcification occurring in constrictive pericarditis, in a hydatid a goitre (X ray 90) or round tuberculous lesions. In most cases a probable, but not positive, diagnosis can be made. The tumour might still prove to be a malignant thymoma, or the comparatively innocent dermoid a dangerous teratoma. Clearly there is everything to be said for the early removal of such things while they still can be simply and safely shelled out, and before complications render their excision difficult.

Exploratory puncture should never be done lest it carry infection or, worse still, malignant cells into the pleural cavity. Early exploratory thoracotomy is indicated, and it is best carried out by a standard postero-lateral incision through the fifth left interspace. The lung is pushed away the mediastinal pleura incised anterior to the phrenic nerve, the tumour inspected and its nature confirmed. It is important to make sure it is not a thymoma or even part of a large retrosternal goitre whose vascular attachments are out of reach. Surgical removal is usually easy before infection, and consists simply in dissecting the tumour from its surrounding structures, no large blood vessels usually being encountered, but after infection it can present great difficulties because of dense and tough adhesions to the parietes and to the origins of the great vessels rendering sharp dissection hazardous but necessary. Malignant change in the tumour may still further complicate the situation, but the aim of surgery should always be complete excision and never the marsupialization once practised.

Pericardial coelomic cysts

These fairly common cysts probably arise by being pinched off from the pneumato-enteric recess of the pleuro-peritoneal cavity during the formation of the primitive diaphragm. They are always situated anteriorly closely related to the cardiophrenic angle, and 70 per cent. of them are found on the right side. Thin-walled, lax, and lined by a single layer of endothelial cells, they contain fluid closely resembling that found in the pericardium, and whose crystal clarity contrasts with the yellowish hue of most other body fluids or cyst contents, and earns for them the alternative title of *Spring water cysts*. Some indeed have a pedicle attaching them to the pericardium, or even a narrow communication with it so that fluid can be expressed from one to the other, the cyst being actually a diverticulum. Most, however have no such attachment, and are either loosely

move with respiration, or fall away from the chest wall with the lung unless they are already invading it.

Although they undergo degenerative changes, with myxomatous, cystic, haemorrhagic and xanthomatous areas, malignant change in them is quite exceptional

2 *Neuroblastomas*

Neuroblastomas occur in small infants and resemble the foregoing in position and origin. They are, however, essentially malignant, and surgical removal must be followed by high-voltage irradiation of the area. Although the ultimate prognosis is grave, recurrence and metastases are sometimes delayed for years. One of my patients, a child of two, who presented with paraplegia in the same way as the case already described, and whose spinal extension was removed by my colleague Mr. D. W. C. Northfield, remains alive and well four years later (X-ray 101). A remarkable and unexpected feature of these tumours in view of their malignancy is that they all show a fine, diffuse speckled calcification on X-rays.

3 *Neurofibromas associated with von Recklinghausen's disease*

Diffuse, uncapsulated or circoid neurofibromas occur fairly commonly in the chest as local manifestations of multiple neurofibromatosis. A familial history, neurofibromas elsewhere, and skin pigmentation help diagnosis. They are particularly prone to malignant change, about a quarter of all neurogenic sarcomas occurring in patients having the stigmata of von Recklinghausen's disease.

These neurofibromas are, of course, not confined to the posterior mediastinum, but are found anywhere on the course of intercostal nerves, beading and tortuously enlarging them. Should the phrenic or vagus nerves be involved, a chain of lobulated tumours are produced down one or both sides of the mediastinum, resembling metastases in lymph glands, and even causing some degree of mediastinal obstruction.

4. *Solitary neuromas or neurofibromas on segmental nerves*

Solitary small neural tumours connected with the lower cords of the brachial plexus or with an intercostal nerve occasionally cause diagnostic problems. They are also subject to sarcomatous change.

Operation

Surgery is indicated in all cases of diagnostic doubt, but is mainly

directed to the removal of the common ganglioneuromas. This usually presents no difficulty and is performed transpleurally through a postero-lateral intercostal thoracotomy. At first they seem firmly fixed to the chest wall but as soon as the parietal pleura over them is incised they can be rapidly shelled out, being attached only by a short pedicle in the paravertebral gutter. Tumours with an extension through an intervertebral space into the spinal canal call for prior neurosurgical exploration, and neuroblastomas should be removed with as much surrounding tissue as possible to ensure extirpation. Neurofibromas connected with von Recklinghausen's disease should, of course, be left alone unless they are causing symptoms or show signs of rapid enlargement but solitary neural tumours require removal both because their identity remains in doubt and to avoid later malignancy. If accessible they can sometimes be shelled out of an intercostal space without the pleura being opened but every effort should be made to dissect them away from the accompanying nerve sheath, especially if it is a contributor to the brachial plexus, without damaging the nerve itself.

Enterogenous cysts

Bronchogenic and enterogenous cysts have a similar origin for both arise, the former at one remove, as diverticula pinched off from the primitive foregut during foetal development. Enterogenous cysts however are lined by gastric mucous membrane, and their subsequent behaviour depends entirely on how much acid or enzymes are secreted by it. As one would expect, they are closely related to the oesophagus and are therefore found posteriorly in nine cases out of ten upon the right side, presumably because of the presence of the aorta on the left (X ray 102). Most have been described in infants under one year.

If the mucosa is active, and secretes a normal gastric juice with all its components, the cyst resembles a closed accessory stomach and is probably incompatible with life because it rapidly distends, bleeds, perforates into nearby structures, or causes death by interfering with respiratory function. Under such circumstances its wall is as thick as that of the stomach and densely adherent to the perietes. A number of instances of peptic ulceration and of penetration of bronchi are recorded. In infancy cough, dyspnoea and cyanosis due to the cyst's bulk, or to collapse of the adjacent lung, may precede or accompany haemoptysis, haematemesis or melaena. Once a fistula is established a fluid level is seen across the X ray shadow revealing its cystic nature. Should aspiration unwisely have been attempted the

presence of hydrochloric acid or pepsin confirms the diagnosis. Removal is life saving.

If the mucosa secretes little but mucus, on the other hand, the cyst remains silent and persists into adult life. Under these circumstances the wall is much thinner, and removal is likely to precede diagnosis which depends upon the recognition of gastric mucosa.

Meningiomas and meningoceles are rare, but also lie posteriorly in the chest and are most likely to be mistaken for neurofibromas. Some are associated with widening of intervertebral foramina and might be diagnosed by myelography.

THE CENTRAL MEDIASTINUM

Tumours of lymphoid tissue

The mediastinum is rich in lymph glands, especially in its central parts surrounding the lung roots, in both pulmonary ligaments, and on either side of the trachea. In addition, it is traversed by the thoracic duct which receives drainage directly from the oesophagus and lungs as well as from below the diaphragm. All this lymphoid tissue is subject both to localized tumours and granulomas, and to generalized lymphatic disorders.

Of these much the commonest is *tuberculous adenitis* associated with primary tuberculous lung infection, although it is usually transitory and demands no treatment. Temporary enlargement also accompanies whooping cough and the bronchopneumonic complications of measles. It may, however, be associated with collapse of a pulmonary lobe, due to external pressure on the bronchus, and in later life a craggy mass of breaking down tuberculous glands is sometimes mistaken for a growth. Such glands are always densely adherent to neighbouring blood vessels and bronchi, rendering dissection difficult. They may ulcerate into a bronchus causing severe haemoptysis, or discharge calcified material that is then coughed up as a broncholith or 'lung stone'. Evidence of pulmonary tuberculosis, cervical adenitis, and radiologically visible calcification in the mass assist diagnosis.

Metastatic deposits

Metastatic deposits are exceedingly common, especially from lung cancers, and they are sometimes more conspicuous than the primary growth. They are most easily seen on the right side of the superior mediastinum and it should be remembered that this area receives drainage not only from the right lung but to some extent from the

lower lobe on the left side across the carinal glands. Similar deposits on the left infiltrate the recurrent laryngeal nerve causing vocal cord paralysis and permanent hoarseness, and on either side they may produce phrenic palsies with paradoxical diaphragmatic movement dysphagia or cervical venous distension. All these are therefore signs of absolute inoperability in patients with cancer of the lung.

Boeck's sarcoidosis

This is a disease of unknown aetiology having some relationship to tuberculosis manifested in its earlier phase by bilateral enlargements of the hilar and mediastinal lymph glands. Certain identification depends on histological changes in the glands.

Lymphosarcoma and lymphoblastoma

These occur the former mainly in young adults, the latter in children. Both present smoothly lobulated massive shadows bulging out from the mediastinum. These together with *lymphadenoma*, are practically the only mediastinal tumours which respond rapidly to deep X rays (though this is not always the case with lymphoblastomas) and irradiation therefore provides a useful diagnostic test. In the case of Hodgkin's disease, which is not infrequently localized at first to the mediastinum, the Pei-Ebstein syndrome and blood picture also assist diagnosis. None of these conditions calls for surgical interference beyond, possibly biopsy of accessible lymphatic glands and all are ultimately fatal.

Lymphangioma, or cystic hygroma

This is a rare mediastinal tumour. It arises as a congenital malformation of lymphatic vessels, is unencapsulated multilocular intimately connected with surrounding structures, and its lymph-containing spaces are lined by flattened mesothelium. Removal can be a formidable procedure as it is supplied by very large aberrant blood vessels. Diagnosis prior to thoracotomy is impossible (X ray 103)

Bronchogenic cysts

Bronchial cysts probably originate as a 'pinching-off' process from one of the outgrowing lung buds, and when this takes place proximally the cyst is found in the central mediastinum close to the carina or attached to it by a stalk. More peripheral ones are described in the chapter on Cysts of the Lung. All are lined by cuboidal or columnar epithelium, contain mucus and have walls in which bronchial elements such as cartilage, elastic tissue and smooth muscle

are found Very large cysts are sometimes seen in infancy and cause respiratory distress and cyanosis; but most remain symptomless unless complicated by secondary infection, when they may give rise first to obscure fever and ill health and later rupture into a bronchus or the pleural cavity. Such fistulae never heal, the cyst thereafter behaving as a chronic abscess.

Radiologically an unruptured cyst cannot be distinguished from a rounded solid tumour (X-ray 104), but once a fistula is established and a fluid level appears, it can generally be recognized by its very fine regular wall Even so, it may resemble gut in a diaphragmatic hernia, a staphylococcal abscess, an infected hydatid, or even a breaking down carcinoma

Surgical removal is planned through whichever side of the chest affords best access to the cyst, and removal presents no great difficulty except that aberrant vessels are sometimes closely related to it, and care must be devoted to closing any bronchial fistula.

Rare tumours

Lymphangioma, lipoma, meningeoma or meningocoele have already been mentioned *Haemangioma* of the mediastinum may be single and encapsulated, in multiple foci as part of generalized congenital telangiectasis, or frankly malignant At least 17 instances are recorded, of which 8 were malignant A vascular bruit may be audible over them, but this is common to all very vascular tumours. *Leiomyoma*, an innocent growth of the smooth muscle of the oesophageal wall, may occasionally complicate the already difficult differential diagnosis of mediastinal neoplasms, as may *parathyroid adenoma*, with which are associated skeletal changes, raised serum calcium and generalized osteitis fibrosa; and plasmocytosis, single or multiple, innocent or malignant; fibroma; chondroma; myxoma; and xanthoma.

DIAPHRAGMATIC HERNIAS

CONGENITAL hernias of the diaphragm on the whole are rare occurring through defects left in the structure during its complex development and are chiefly important because of the risk they involve, especially in infants, of intestinal obstruction or strangulation. Acquired hernias on the other hand are of two sorts, those due to trauma which are not common and sliding hernias through the oesophageal hiatus which are met with in great and increasing numbers and are one of the commonest causes of dyspepsia.

CONGENITAL HERNIAS

The primitive coelom is partitioned off into the separate peritoneal and pleural sacs by the fusion of a number of diverse elements the septum transversum of the ventral mesentery portions of the third and fourth cervical myotomes, which retain their innervation from the third and fourth cervical nerve roots in what is later to be the phrenic nerve ingrowths from the lateral myotomes the dorsal mesentery and the pleuropertitoneal membranes. The complexity of such varied contributions leaves a great deal of room for defects to occur, and it is perhaps surprising that congenital hernias are not more common. They are found chiefly in four situations anteriorly through a gap called the foramen of Morgagni posteriorly through another called the foramen of Bochdalek through an abnormal oesophageal hiatus and through a deficiency of the central tendon of the diaphragm. Often they remain undiagnosed until symptoms occur or are recognized after a chest X ray has been taken for some other reason, but sometimes impairment of physical signs in the chest or the presence of bowel sounds in it leads to their detection.

1 *The Foramen of Morgagni* is a D-shaped deficiency at the point where the internal mammary artery normally pierces the diaphragm to reach the abdominal wall, its flat anterior boundary being the back of the sternum or lower costal cartilages in front, and its curved boundary formed by the arching muscle of the diaphragm behind (X ray 105). Because it is anterior transverse colon or omentum are the structures usually herniated through it and whereas the former is easily recognized, the latter resembles radiologically a solid

tumour. The defect, which may be bilateral, is repaired by anchoring the free edge of the diaphragm to the anterior chest wall by interrupted floss silk sutures.

2 *The Foramen of Bochdalek* represents a continued patency of the pleuro-peritoneal canal and is, therefore, always posterior in position. As the liver protects the right side, hernias usually occur on the left, and contain small intestine.

3. *The oesophageal hiatus* is sometimes abnormally patulous and permits a large herniation of the stomach into the chest. Unlike the much smaller acquired type of sliding hiatal hernia, this congenital kind is relatively symptomless. The gastric fundus rotates and presents in the right chest where it resembles radiologically a thick-walled cavity containing a fluid level. It is this type of hernia which is rather pointlessly called 'partial thoracic stomach'.

It is true, however, that in newborn infants a much smaller sort of hiatal hernia occurs, indistinguishable from those seen in the adult and often, in marked contrast to large protrusions, associated with severe symptoms. These principally consist in regurgitation at the beginning of feeds or projectile vomiting, in which all the milk, with much mucus, is returned, sometimes streaked with changed blood. Although the child seems hungry and takes food well, weight is rapidly lost and eventually the infant becomes dehydrated. The symptoms are often thought to be due to intestinal obstruction, cerebral irritation, or haemorrhagic disease of the newborn, and much time may be lost in treating children for 'cyclical vomiting' or 'acidosis', or even pyloric stenosis, if the true explanation is missed.

Acid reflux, as in the adult, causes intense oesophagitis, and if this is not promptly checked leads to stricture formation, a very serious complication which seems to occur more quickly in infants than in adults. The so-called 'congenital short oesophagus' which used to be described is simply an oesophagus connected with such a sliding hernia, in spasm due to oesophagitis, or permanently shortened by the resulting cicatrization. If nursing in a continuously upright posture is at once begun (the child being kept up at night in a special harness), the great majority lose their symptoms, some quite quickly, but others lingering on for a year or two with longer and longer remissions, the attacks returning only during illnesses or if the patients are allowed to lie flat at night. Weaning, and a change from fluid to a more solid diet is also beneficial.

Should response to this treatment not be prompt it is imperative that the sliding hernia be repaired surgically at once, and acid reflux



105

X-ray 105. Herniation of bowel through a patent foramen of Morgagni. Note the position of the hernia immediately behind the sternum.



106

X-ray 106. Eventration of the left diaphragm in an elderly man. The fluid level is in the stomach. The disastrous effects of this on the left lung are obvious. (See also X-ray 107.) He was very breathless and coughed up 2-3 oz. of pus every day. Repair of the eventration in childhood would have saved the lung.



107

X-ray 107. Bronchogram of the lung seen in X-ray 106. There is severe bronchiectasis in the left lung. The appearance of 'rat tailing' in the right lower lobe bronchi is typical of chronic bronchitis.



108

X-ray 108. Eventration of anterior part of left diaphragm in young boy. It moved paradoxically on screening, and contained only of pleura and peritoneum. Normal diaphragmatic muscle was sewn to close defect. Thereafter the whole area moved normally and boy was thus saved fate of patient shown in X-rays 106 and 107.



109

X ray 109 Traumatic hernia of left diaphragm. Stomach lies in chest, compressing left lung and displacing mediastinum to right. Ribs 5, 6, 7, 8 and 9 are fractured, and spleen (lying just behind 10th rib) is ruptured. At thoracotomy stomach was replaced, spleen removed, diaphragm repaired, lung re-expanded.



110

X ray 110 Typical rather large, sliding hiatus hernia. One of the commonest causes of dyspepsia and much the commonest type of diaphragmatic hernia. Cardia is well above sphincter of right diaphragmatic crus: a pouch of stomach lies in the chest and there is nothing to prevent acid reflux into gullet. Patient had severe oesophagitis.



111

X ray 111 An example of para-oesophageal hiatal hernia. The cardia is not much displaced, but the fundus of the stomach has rolled up through the hiatal orifice and now lies beside the lower third of the oesophagus. This type of hernia is much less common than the sliding variety, and may not be associated with acid reflux. Compare this with X ray 110.



112

X ray 112 A large swallow in a patient suffering for many years from dyspepsia due to a small hiatal hernia. There is intense oesophagitis and spasm of the lower end of the gullet, and the X-ray appearances are almost those of oesophageal cancer. The patient was unable to swallow solids. Now compare X ray 113.



113

X-ray 113. A second barium swallow taken three months after repair of the hiatal hernia causing the irregular structure seen in X-ray 112. The patient was able to swallow freely within a week or two of operation and has never had recurrence of symptoms.



114

X-ray 114. A cavity with fluid level at the right base. Patient expectorated much pus and was referred as case of lung abscess. A barium meal (see X-ray 115) shows cavity to be stomach herniated through a wide hiatal orifice. This defect in diaphragm rendered both lower lobes bronchiectatic and pus came from them. The fluid level is in the stomach.



115

X-ray 115. A barium meal given to the patient whose chest is seen in X-ray 114 shows the stomach to be in the chest. Repair of the hernia greatly diminished breathlessness and sputum soon decreased.



116

X-ray 116. Safety-pin lodged in infant's gullet. Attempts made to remove pin by pulling on its head resulted in perforation. In the X-ray right pyo-pneumothorax is visible. If the power not the head, had been drawn into the oesophagoscope, removal would probably have been successful. Pus was removed at right thoracotomy and oesophagus repaired.



117



118

X-ray 117 New born infant with commonest type of tracheo-oesophageal fistula. Upper blind end of gullet is outlined by Lipiodol. Note its size, contrasting with lower end which communicates with trachea. Note also gas in gut proving fistula between gullet and wind-pipe exists.

X-ray 118 An innocent stricture in the lower third of the oesophagus secondary to acid reflux caused by a small hiatal hernia. Dilatation would have made this worse, and it was treated by oesophago-gastrostomy, most of the acid-bearing part of the stomach being removed. This is the consequence of not repairing hiatal hernias betimes.



119



120

X-ray 119 A large pharyngeal pouch outlined by barium. Note how it displaces the oesophagus forward so that both food and instruments are much more likely to enter the pouch than the gullet.

X-ray 120 The typical appearances of cardiospasm outlined by barium. The oesophagus is enormously distended, and the hold-up is at the cardia.

checked, irrespective of the age of the child. Failure to do so inevitably invites stricture formation. This is rare before six months, but overtakes perhaps 10 per cent. of those affected. In some, repair of the hernia and dilatation may suffice but in many an oesophago-gastrostomy with removal of the acid secreting part of the stomach may have to be performed.

It is difficult to say whether such hernias are truly congenital or not, but as they are seen so early in life they presumably are the consequence of some inborn defect of the hiatus or of its mechanism.

4 There is sometimes a deficiency of *the whole central tendon of the diaphragm*, when bowel on the left, liver on the right, present through it, the latter resembling a solid intrathoracic tumour. When the gap is very great abdominal viscera fill the left chest and the diaphragm is represented only by a cuff of muscle around the thorax.

It is not the case that congenital hernias can always be distinguished by having no sacs for whereas this is usually true they may be covered by a sheath of peritoneum or of pleura. An intermediate stage is that of *eventration of the diaphragm*, the whole or a part of one dome (almost always the left) protruding as a high arch into the chest, and usually filled by the stomach whose wall makes the eventrated part appear radiologically as thick as the rest of the diaphragm (X-ray 106). This is not in fact so, for it consists of a wide-mouthed aperture covered only by tenuous fibrous tissue and pleura. As no muscle is present in it at all this area usually moves paradoxically with respiration. This has led to an erroneous belief that eventration is due to idiopathic phrenic paralysis. All cases of eventration should be explored and the defect repaired (X ray 108).

A man aged 68 complained of increasing breathlessness. On questioning he admitted that all his life he had found it difficult to keep up with other people but this had not worried him until quite recently. He had had varying amounts of purulent sputum for many years. On screening, a large eventration of the left diaphragm was seen, the eventrated portion moving up when the patient sniffed the normal part of the diaphragm, down. The whole of his left lung showed gross cystic bronchiectasis the consequence of his being unable to cough effectively due to the paradoxical movement (X ray 107). His age and general state did not permit surgical treatment, but had his condition been recognized in youth, the eventration could have been repaired by sewing normally functioning muscle across the gap preventing subsequent collapse and irreparable damage to his lung.

The effect of any substantial diaphragmatic defect on the lung is

similar, for the diaphragm is the most mobile, and consequently the most important, part of the chest wall. Breathlessness is a symptom—sometimes the only symptom—of a sizeable diaphragmatic hernia of any kind, a large hernia having much the same effect as a sucking pneumothorax (see page 72).

The protrusion of bowel through a congenital diaphragmatic defect may lead to strangulation, with all the characteristic signs of intestinal obstruction, complicated by cyanosis and breathlessness if much intestine is in the chest. Surgical intervention is life-saving. A tube is passed to aspirate the stomach contents and to diminish its bulk, and the hernia is approached directly by thoracotomy. Reduction is often difficult owing to bowel distension, and sometimes to the fact that the bowel has always dwelt in the chest and never in the abdomen. The table head should be tilted up to help reduction, but in cases where it is otherwise impossible a laparotomy incision is made over which the skin alone is sutured, leaving a ventral hernia to be repaired later. Diaphragmatic defects are closed by direct suture if they are small, by fascial or floss silk grids if large.

Congenital hernias which do not cause symptoms in childhood persist into adult life to cause dyspnoea (as the thorax becomes less mobile and the patient more reliant upon an efficient diaphragm), or vague abdominal discomfort; or to be discovered on routine radiography (when hernias of liver or omentum may resemble intrathoracic tumours). Unfortunately, however, it is much more usual for breaking-down growths or other abscesses to be mistaken for diaphragmatic hernias. Abscesses are common; hernias (apart from hiatal ones) rare.

TRAUMATIC HERNIAS

In war-time most of these are due to penetrating thoraco-abdominal wounds, in peace, to non-penetrating, crushing injuries sustained in traffic accidents which rupture the central tendon of the left dome of the diaphragm leading to extrusion of the stomach and intestines into the chest.

I was once called to see a man of 19 who had been thrown from his motor-cycle two days previously, suffering fractures of one femur, a radius and ulnar, and four ribs on the left side. He was blue and breathless and thought to have a left haemothorax. An X-ray of his chest showed what at first seemed to be an enormously high left diaphragm, with the pleural cavity above it quite opaque, and below it a pneumoperitoneum (X-ray 109). In the midst of the latter the spleen was clearly visible broken into three pieces. At operation I

found that the left diaphragm had been split across from side to side, the muscle retracting to the periphery so that it was quite difficult to see. The chest was occupied by almost the whole of the stomach, together with a good deal of blood and air which had leaked from the collapsed and torn lung and in the abdomen, not bleeding at all, lay the spleen in three pieces. After splenectomy through the chest, the stomach was replaced, the leaves of the diaphragm sewn together, and the lung re-expanded. Recovery was uneventful.

HIATAL HERNIA

Of all the out patient departments of a large hospital that dealing with chronic dyspepsia is perhaps the most dispiriting. There sit the rows of old familiar drooping faces there are the well-thumbed record sheets covered with alkaline prescriptions whose nature varies a little with fashion but not much, relapsing at last into *Rep. mist.*

Rep. mist. From it occasional sorties are made to the X ray department for barium meals and cholecystograms or to a surgical ward for partial gastrectomy or to the psychiatrist with a note 'functional dyspepsia'. Among the many thousands with serious indigestion attending such clinics less than a third have a demonstrable peptic ulcer, and about a quarter have at some time or other rightly or wrongly been diagnosed as functional dyspepsia. Should that fair fat woman of forty with three children, whose gall bladder is so normal, be sent to join them? Before she is, or before *Rep. mist.* is inscribed again, it is worth while asking some special questions. For it has been estimated that between a quarter and a third of all patients complaining of dyspepsia suffer from acid reflux into the oesophagus and in the great majority of these, though not in all, a sliding hernia of stomach through the oesophageal hiatus can be demonstrated radiologically. It is responsible for the symptoms can be cured surgically and is thus by far the commonest variety of diaphragmatic hernia, as well as being one of the commonest causes of dyspepsia. The smallest hernias often cause the worst symptoms.

The lower end of the oesophagus has no intrinsic sphincter like the anus, but is controlled instead by the muscles of the right crus of the diaphragm which are arranged in a sling round it, and function in the same way as does the pubo-rectalis muscle sling around the rectum. Contraction of this loop increases the angle at which the oesophagus joins the cardia and shuts it off like a pinch-cock. In addition, and much less importantly, some of the fibres decussate around the lower end in scissor fashion so that in contracting they help to close it. If this mechanism is intact, reflux of gastric contents into the

oesophagus cannot occur; but if for any reason the cardia becomes displaced so that it lies above the muscle sling, in a hiatal hernia, there is no mechanism to prevent reflux (X-ray 110). The stomach may be pushed through by intra-abdominal fat, or by pregnancy; or it may be pulled through by longitudinal spasm of the oesophagus occurring reflexly from vagal irritation due to gall-bladder disease, or duodenitis, or reflux oesophagitis itself, so that a vicious circle is formed; or the musculature of the hiatal sling may be deficient, allowing the stomach to slide up through it when the patient goes to bed or bends down. Usually all these things happen together, supplementing and perpetuating each other, so it is not surprising to find hiatal hernias commonest in fat middle-aged women of poor musculature who have had several pregnancies; although, as we have seen, they can also occur in new-born babies

Most of those who suffer from an hiatal hernia have had an average of three previous diagnoses, in spite of the symptoms being characteristic; but patients do not volunteer an account of them unless specifically asked; and as most have been attending clinics for many years they seldom are asked. Furthermore, as the cardia is merely protruded through a patulous hiatus, the hernia that it forms is a sliding one with only a partial, frontal sac, and in most cases it reduces spontaneously when the subject stands erect. It is therefore essential that during examination with barium the patient bends down to touch the toes or is placed in a Trendelenburg position so that the protrusion and accompanying reflux can be demonstrated: it is not enough to ask the radiologist merely for a barium swallow or meal without indicating the reason for it.

The dyspepsia associated with hiatal hernia is a recurrent burning pain, high in the epigastrium or behind the lower end of the sternum not directly related to food, but rather to posture, coming on when the patient bends down or goes to bed. Sometimes it is felt precordially and radiates to the back, the neck, or the left arm. As bending is often connected with effort, such as scrubbing floors, lifting weights, or gardening, this pain may be confused with angina pectoris. Indeed the nerve paths of both types of pain are the same, experimental distension of the stomach decreases coronary flow; and electrocardiograms taken when a hernia is present may suggest coronary insufficiency, yet be normal when it is reduced. (Nazum found a hiatal hernia present in 25 out of 100 patients diagnosed as having angina pectoris; but in only 12 per cent of 957 given barium meals for other reasons.)

With the pain go flatulence, 'heartburn' and acid regurgitation

without nausea, initiated by bending, or coming on in bed. These symptoms, which are caused by the reflux of acid into the lower end of the oesophagus, are usually relieved by standing upright or by using several pillows at night. They are aggravated by fatigue and by emotional upsets.

In the much less common paraoesophageal type of hernia, the cardia remains in its proper position, but the fundus of the stomach rolls up through the hiatus to lie beside the oesophagus (Fig. 33) As reflux does not then occur the only symptoms to which it gives

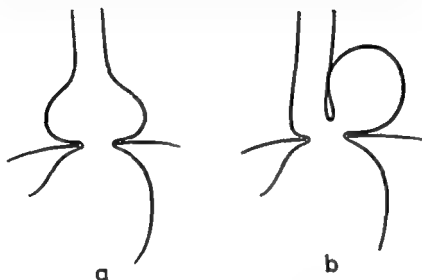


FIG. 33

The difference between the common sliding type of hiatus hernia (a), and the much less common paraoesophageal type (b) Note that in the latter the cardia is little, if at all, displaced.

rise are substernal discomfort, belching, and intermittent dysphagia (X ray 111)

Not all patients with reflux develop oesophagitis but the higher the acidity the more likely is it to occur. The mucosa of the lower third of the oesophagus becomes reddened, swollen and excoriated and bleeding from it may result in insidious anaemia and the presence of occult blood in the stools. Frank haematemesis or melaena are rare. The oesophagitis causes in turn much reflex spasm of the musculature, that of the circular fibres resulting in some dysphagia, and of the longitudinal coat in a contracture which tends to draw the hernia still higher in the chest and so to aggravate the deformity. Later, true peptic ulceration appears, and the pain and bleeding are

likely to become worse. There is then a marked inflammatory reaction in the mediastinal tissues related to the oesophagus, which becomes fixed and adherent to the pleurae, the diaphragm, the pericardium and the aorta. In some instances perforation occurs into the pleural cavity with a resultant empyema, and, more catastrophically still, such ulcers have been known to penetrate the heart or the aorta. It is in the presence of ulceration and irregular spasm of the lower oesophagus that the condition is often mistaken for a carcinoma. A frail old lady of 68 who had lately been losing weight and had difficulty in swallowing was sent for oesophago-gastrostomy, having been diagnosed, after a barium swallow, as having oesophageal carcinoma. She gave a history of having had 'indigestion' for 25 years which had been treated by diet and with a great variety of alkalis, relieving it to some extent but never altogether. Lately she had found solid food difficult to swallow. A barium swallow revealed an irregular stricture at the lower end of her oesophagus (X-ray 112). As soon as the question was put to her she said that gardening, of which she was very fond, made her much worse and that acid came up into her mouth when she bent down to weed. The barium swallow was repeated and a small hiatal hernia demonstrated. Oesophagoscopy revealed an intense oesophagitis with active ulceration. Thoracotomy was performed, the hernia reduced, the hiatus repaired, and the vagi divided to reduce acidity. She made a rapid recovery, could soon swallow freely, and now spends most of her time gardening. Although she had had constant indigestion for 25 years, she has had none since the operation 5 years ago, takes no alkalis, and eats what she chooses (X-ray 113).

Long-standing oesophagitis and ulceration lead inevitably if untreated to fibrous stricture; and it is probable that the great majority of innocent strictures of the lower end of the oesophagus are so caused (X-ray 119). As the stricture tightens dyspepsia and acid regurgitation diminish, giving way instead to intractable dysphagia. To dilate such a stricture is to restore the reflux: the only satisfactory treatment is oesophago-gastrostomy. Although this would appear to restore the original defect, most of the acid-secreting portion of the stomach, along the lesser curve, is removed, both vagi are inevitably cut, and neither symptoms nor oesophagitis usually recur.

Oesophagoscopy is an essential step in the investigation of all cases of hiatal hernia, in order to confirm the diagnosis and exclude carcinoma, and to observe the distance from the gums at which gastric mucosa is encountered, the degree of spasm and oesophagitis, and the presence of ulceration or of a stricture.

The oesophagoscope usually passes easily through into the stomach, but if there is much spasm due to inflammation this may not be possible (as well as being unwise), and a large gum-elastic bougie is passed instead. When the mucosa is much inflamed, or an active ulcer present, but no fibrous stricture the best treatment is to prevent any further oesophagitis due to reflux by restoring the normal anatomy as promptly as possible. In most cases repair is followed by complete relief of symptoms, and a subsequent barium swallow will prove that the oesophagus is normal in appearance the hernia cured, and that no reflux can be produced.

Finally it must be remembered that in patients with low acidity dyspepsia and reflux are not always the chief symptoms. A man of 48 was referred after a lung abscess had been diagnosed clinically and apparently confirmed radiologically.

He was coughing up 4 to 6 ounces of thick pus every day and was very short of breath but on questioning it transpired that this had been going on for a year or more. His X ray showed what looked like a thick walled abscess with a fluid level in it, and after postural drainage the level disappeared (X ray 114). Further investigation showed the abscess to be in fact a herniation of stomach into the right chest (X ray 115). Because of the large deficiency at the hiatus function of both sides of his diaphragm was impaired so that the lower lobes of both lungs had become grossly bronchiectatic, and from them came the pus he coughed up. As soon as his hernia was repaired he became much less breathless and was able to cough effectively.

Operation

The left chest is entered through the bed of the seventh rib and the lung packed upwards. As soon as the layers of the pleura which form the inferior pulmonary ligament are divided to expose the lower end of the oesophagus, the cardiac portion of the stomach is seen bulging through the hiatus, partially covered by peritoneum. This is dissected free and the lower end of the oesophagus mobilized sufficiently to make reduction of the hernia easy and this is very seldom impossible, the only difficulty arising when real shortening has taken place due to scarring. The central tendon of the diaphragm is incised for a short distance to afford access to the stomach and under-surface of the hiatus, and the reflection of peritoneum on to it is cut (Fig. 34a). The muscular sling of the crus is then clearly visible, and a forceps is passed up between it and the stomach to grasp a plastic tape passed round the oesophagus to draw it down

wantonly and for ever sacrificed In a younger patient the consequences may not be serious, but with advancing years the victim pays a growing price in breathlessness, and probably in lung infection *There can be no justification for robbing a man of his breath to cure his indigestion.*

PART TWO

SURGERY OF THE OESOPHAGUS

DYSPHAGIA

WHEN the oesophagus is diseased difficulty in swallowing is almost always the symptom which sends the patient to his doctor. It is a symptom which usually has an organic basis, and calls for vigorous investigation and accurate diagnosis. The first and perhaps most important step in such an investigation is to elicit, and patiently listen to a full history of the trouble. A distinction must be made between difficulties in eating such as may occur in any local disease of the mouth, the tongue or the pharynx, and difficulty in the act of swallowing itself. The former may be caused by a quinsy, by bulbar palsy or by a growth of the tongue and is *disordered deglutition*, not dysphagia.

The history is sometimes long, going back for many years and suggesting an innocent rather than a malignant cause but it must not be forgotten that carcinoma may involve a gullet already affected by cardiospasm or by acid reflux. Childhood difficulties a record of swallowed corrosives, or foreign bodies or of chronic dyspepsia are all relevant. If fluids were first held up rather than solids, muscular spasm is suggested, and is made more likely if there have been complete remissions. But the existence of remissions does not exclude the possibility of growth, especially one near the cardia, for abrupt dysphagia due to muscular spasm may be the first symptom of a carcinoma, and may later remit for quite a long period before it returns. Later in the disease persistent dysphagia sometimes ceases because the lumen is held open by rigid growth encircling its wall.

An eminent civil servant enjoying the best of health was dining at Clarges with a thoracic surgeon. In the middle of the meal he excused himself and left the table. As he was gone some time the surgeon followed him and found him trying to retch. 'Something I ate' he explained 'stuck just there—pointing to the lower end of his sternum—and would go neither up nor down. Such a thing has never happened to me before. He was admitted to hospital for investigation by which time he had no difficulty whatever in swallowing. An oesophagoscopy revealed a carcinoma just above the cardia, later proved to be inoperable.

Sometimes the leading symptoms are *pain* rather than difficulty

or *regurgitation*, in which undigested food is returned without nausea or effort, mixed with frothy mucus, and alkaline rather than acid in reaction. Such a process is often described as 'vomiting' but close attention to the history reveals its true nature. A *productive cough*, nearly always precipitated by lying down at night, and caused by the aspiration of food particles from a distended oesophagus or pouch into the lung, frequently accompanies chronic disorders and is often the symptom for which the patient seeks advice.

The next step in investigation is for the surgeon himself to watch the patient drink and take food, as it is only in this way that a true assessment can be made of the difficulties and severity of the trouble. The length of the pauses between mouthfuls, the size of the portions taken, the frequency with which drinks are needed, the manœuvres resorted to, the gurgling noises which accompany pouches, are all carefully observed.

A general examination may confirm that weight has been lost; but this can be due to inadequate nutrition as well as to cancer. Foul breath is common in many oesophageal disorders, but is particularly noticeable in the presence of a fungating neoplasm. Hoarseness due to recurrent laryngeal palsy; malignant glands in the neck, an enlarged and irregular liver; a craggy mass of gastric neoplasm below the left costal margin, better felt if the patient is turned on his right side, neoplastic nodules on the pelvic peritoneum felt on rectal examination—all must be sought, but all are often absent.

Clinical and radiological examination of the lungs may reveal chronic pneumonitis, collapse, pleural effusion or even lung abscess due to aspiration of foods. Bronchoscopy is indicated if any of these lesions is present, or if coughing immediately after taking food suggests the presence of a malignant oesophago-bronchial fistula. Although carcinoma of the oesophagus is the commonest *intrinsic* cause of dysphagia, mediastinal involvement by carcinoma of the lung is undoubtedly the commonest *extrinsic* cause. Such a growth must be excluded, and distinguished from the pulmonary complications of oesophageal disease. Dysphagia in cancer of the lung is a sign of inoperability, but calls for palliation with deep X-rays.

A blood count to exclude the hypochromic anaemia of the Plummer-Vinson syndrome, and anaemia secondary to growth or ulceration, is essential. Occult blood in the stools does not necessarily spell malignancy, but may be found whenever the mucosa is ulcerated.

Most useful, and generally conclusive, is X-ray screening while the

patient takes a barium drink, and both the peristalsis of the oesophagus and the nature of any deformities or organic obstructions are noted. *Once a patient has symptoms it is rarely that a barium swallow does not reveal their cause*

Lastly, oesophagoscopy must be performed.

OESOPHAGOSCOPY

DIRECT inspection by the passage of an oesophagoscope is indicated in every case of dysphagia even though the diagnosis as established by the history and by a barium swallow seems certain. Not only does it permit a biopsy to be taken when a growth is known to be present, but it sometimes reveals an unsuspected neoplasm superimposed upon a long-standing innocent lesion. Furthermore, at the lower end of the oesophagus smooth muscular spasm secondary to a cancer may be mistaken for cardiospasm, or conversely a peptic ulcer for growth; whereas at the upper end simple cricopharyngeal spasm looks irregular and resembles malignancy. Only by oesophagoscopy are these doubts finally set at rest and a more accurate idea obtained of the position, the extent and the fixity of a lesion.

As well as its indispensable value in diagnosis oesophagoscopy is used therapeutically in the removal of swallowed foreign bodies, in the dilatation of innocent strictures and of cardiospasm, and in the palliation of some inoperable cancers by the insertion of radon needles or of a Souttar's tube.

The removal of foreign bodies, many of which are not radio-opaque, must be carefully planned. Although it is true that difficulties increase with the duration of the impaction, time should be taken for adequate preparation, and if possible a similar object obtained and trials made to find the best method of extracting it and the most efficient forceps for the purpose. Sharp points or cutting edges are drawn up within the instrument lest laceration produced during attempted withdrawal prove much worse than the original injury, if any there be, caused by the lodgement itself (X-ray 116). If endoscopic removal seems likely to result in further injury, a right thoracotomy is to be preferred. The oesophagus is incised over the object, which can be directly felt, and after it has been extracted both mucosa and oesophageal muscle wall are repaired before the chest is closed.

Simple strictures are inspected and dilated for the first time at oesophagoscopy, but subsequent dilatations are better performed directly by the patient. This, and the use of the Negus hydrostatic bag in the treatment of cardiospasm, are described later.

The oesophagoscope is a much more dangerous instrument than

the bronchoscope and should be employed only by those skilled in its use. Of all foreign bodies which find their way into the gullet it is without question the commonest cause of oesophageal perforation and it is not without reason that oesophagoscopy became known in some quarters as 'sarcophagoscopy'. Instrumentation in the presence of malignancy especially attempts to dilate malignant strictures, often leads to rupture but the commonest site for damage is the posterior oesophageal wall in the cervical region, where hyperextension of the neck arches the cervical vertebral bodies forward and crushes the wall between them and the rigid tube of the instrument. It should be noted that this can happen not only as the beak advances down the oesophagus but after it has passed the danger area. The risk is greatest during long instrumentation and when sharp arthritic osteophytes add to the prominence of the vertebrae.

For general work the best oesophagoscope is the Negus type which has a wide flattish bore and two lamps set obliquely in its walls at the proximal end. It is 45 cm. long, but a shorter one of 35 cm. is used when lesions in the upper part of the oesophagus are to be inspected. Powerful suction and several wide bored aspirating tubes must always be available as well as a selection of biopsy and grasping forceps, gum-elastic bougies, and a hydrostatic dilator bag.

Oesophagoscopy is best performed under good general anaesthesia with intubation. The patient's occiput lies in a bronchoscopy head rest, with the shoulders overlapping the end of the table. Instead of the head being extended as in bronchoscopy it should be somewhat flexed upon the neck, and the neck on the trunk. Not only does this position make the passage of the oesophagoscope easier but it avoids the dangers of hyperextension of the cervical vertebrae.

The instrument is passed into the mouth, usually on one side, and guided downwards with the fingers of the left hand which also prevent it bearing upon the teeth. The epiglottis is identified and lifted forward, and the beak passed into the pharynx behind the arytenoids to the back of the cricoid cartilage. This is the most difficult stage, and it is made easy by the anaesthetist grasping the whole larynx in his hand and gently lifting it bodily forward. The oesophageal lumen at once opens up and can be followed down always under direct vision and aided by frequent aspiration. No force must ever be used and if the instrument does not slide forward easily it is either going in the wrong direction or a stricture has been encountered. Below the level of the aortic arch the head is moved downwards and to the right and there is usually a little forward lift of the beak needed as it passes the cardia and enters the stomach. This point

is recognized by the abrupt change in the appearance of the mucosa and by the reflux of gastric juice. Measurements of any abnormalities encountered are made in centimetres from the upper incisors or gums. In the average adult male the cardia is encountered at 40 cm from the upper gum; it is 15 cm. to the superior constrictor, and 25 cm. to the level of the left bronchus.

If any suspicion of damage arises during oesophagoscopy the patient is given penicillin and a close watch is kept for tenderness, redness or swelling in the supraclavicular region or suprasternal notch. Surgical emphysema as shown by crepitus in these situations is diagnostic of rupture; and X-rays may show air or fluid or both in one or other pleural cavity. There may be fever, and a rising pulse. Sometimes perforation is accompanied by rigidity of the upper abdominal muscles, severe pain and shock. These signs accompany oesophageal tears from any cause. Rare instances of 'spontaneous' rupture are occasionally seen in alcoholics after vomiting, when the rent is usually a longitudinal one of the left postero-lateral wall just above the diaphragm. In all these circumstances food or drink by mouth is at once stopped, the site of injury explored, and the tear sewn up. The lung is then re-inflated, an underwater-sealed drain left in the pleural cavity, and the chest closed. Gastrostomy is not as a rule necessary as fluids by mouth can be started again after 48 hours.

The presence of an aneurysm, or recent swallowing of corrosives are both absolute contra-indications to oesophagoscopy.

CONGENITAL TRACHEO OESOPHAGEAL FISTULA

VERY rarely indeed complete or partial simple webs are found in the infant oesophagus but a congenital anomaly quite frequently encountered is complete atresia of the oesophagus combined with a fistula between its lower segment and the trachea. This deformity is the commonest cause of blueness and choking in the newborn child after its fauces have been cleaned out for the first time. It occurs about once every 2,500 births and is therefore twenty times commoner than ectopia vesicae. It is incompatible with life, but can be cured surgically. Success in treating it is directly proportional to the promptitude with which it is recognized and the child brought to operation, each feed taken after birth loading the scales more heavily against survival.

Both oesophagus and trachea are evolved from the primitive fore gut by two longitudinal ridges which form down each side of it, meet, and so divide it into two tubes. Partial misdirection of the process results in oesophageal atresia, and a fistula persisting between oesophagus and trachea. In 80 per cent. of cases the upper part of the oesophagus ends in a blind pouch closely related to the back of the trachea and extending some way into the superior mediastinum, sometimes almost as far down as the carina, but usually just through the thoracic inlet (Fig. 35). This upper pouch receives any



FIG 35

Varieties of congenital tracheo-oesophageal fistula. Type 1 accounts for 80 per cent. of cases. The next commonest is Type 2 in which the gap between the oesophageal segments is so great they cannot be brought together.

milk or saliva that is swallowed, before they spill over and are aspirated into the lung, where they are likely to cause septic pneumonia, collapse and eventual death by drowning if diagnosis is delayed. This happens even if the baby is fed through a gastrostomy, for saliva is still swallowed and will overflow. The lower segment is narrow and atrophic. It opens into the back of the trachea just above the bifurcation, so that there is direct communication between wind-pipe and stomach. As a result of this the stomach and intestines become distended with air (X-ray 117). Providing the condition is diagnosed quickly, before the lungs are hopelessly damaged and the baby drowned, the two ends of the oesophagus can be joined up and the fistula closed

In the remaining 20 per cent of cases three variants are possible (1) In most there are two blind ends to the gullet, the upper one as before, the lower reaching just above the diaphragm. There is no fistula communicating with the trachea. As the ends cannot possibly be brought directly together this type offers little hope of successful surgery, although an attempt should be made to exteriorize the upper end in the neck and perform a gastrostomy in the hope that later, if the child lives, an oesophago-gastrostomy or jejunostomy can be done. As there is no fistula, *no air can be seen in the bowel on X-rays*. The other types, much rarer, are (2) that in which the fistula is between the upper pouch and the trachea; and (3) that in which both ends of the oesophagus communicate with it (Fig. 35). As with all congenital deformities, other anomalies such as imperforate anus may also be present, and must be looked for, but they are very seldom so severe as to forbid surgery

As a rule a baby is born apparently normal in all respects save that it is blue and choking. Many infants at birth may splutter and go blue; but once their fauces have been wiped out there should be no further trouble—a recurrence of cyanosis, frothing at the mouth and choking, must always bring to the midwife's or the doctor's mind the question 'Has this baby a tracheo-oesophageal fistula?' Such a question may be life-saving, for once a feed is given partial drowning is inevitable. The question can be answered at once by passing a fairly large, lubricated, soft rubber catheter into the mouth and down the gullet. If it stops at about 4 inches, or 10 cm., from the gums and will go no farther, atresia is virtually certain. The child should be hurried to hospital, feeding of any kind forbidden, and the fauces kept regularly aspirated, or carefully wiped dry. Unfortunately prompt diagnosis is exceptional. The majority have already had one or more feeds before choking over the milk, and cyanosis,

arouse misgivings. When put to the breast they always suck hungrily and the severity of their symptoms is redoubled. Nevertheless the airway must be cleared, feeding stopped, and surgical aid sought with all haste.

When the child reaches hospital further aspiration of the airway is carried out if necessary and a radiograph of the chest and abdomen obtained. In particular the presence or absence of gas in the guts is observed. It is not necessary to outline the upper pouch with Lipiodol and the practice is dangerous as spill-over into the lung may occur. The child is prepared for early operation. From this moment on the closest collaboration between surgeon, paediatrician and nurse is essential to success. If much time has elapsed since birth, or if the child has been fed, pneumonia, pulmonary collapse and dehydration add to the difficulties of its care. The bronchial tree is kept aspirated, intramuscular fluids are given and antibiotic therapy begun. Intravenous fluids are avoided if possible as danger lies rather in giving too much than too little. Although there should be no undue delay in surgical repair it is foolish to operate until the child has been got into the best state possible, and six or even twelve hours spent in preparing it often pays dividends, but these hours should be spent in hospital, not at home.

Operation

The chest is opened postero-laterally through the fifth intercostal space and the mediastinum approached directly. No attempt is made to dissect extrapleurally. The azygos vein is cut between ligatures and the trachea and oesophagus exposed. The blind upper pouch is easily found if the anaesthetist passes a polythene tube into it. It is mobilized, especially from the back of the trachea to which it is quite densely attached, and drawn down into the thorax. The much narrower lower end is found lying in the posterior mediastinum and traced upwards until its fistula with the back of the trachea is visible. This is cut free and the hole in the trachea closed with interrupted vascular sutures. Excessive mobilization of the lower end is avoided as far as possible for it involves interference with the blood supply. Whatever length is needed to bring the ends together is gained rather from the upper pouch whose wall is much thicker and more robust. When the two are satisfactorily opposed, without tension the lumen of the upper pouch is opened to match that of the lower. The polythene tube is passed through, and a meticulous anastomosis is carried out using interrupted sutures of the finest silk in one layer through the whole thickness of the walls. The mediastinal pleura

is not closed, as it is better, if fluid accumulates, for it to do so in the pleural cavity; nor is the chest drained, as the presence of tubes greatly complicates the already difficult nursing.

The main postoperative concern is to see that fluid is kept out of the lungs. This demands constant vigilance and frequent aspiration of the fauces and, if necessary, the trachea. It is far more often the cause of death than is leakage at the anastomosis. Suction equipment and an infant laryngoscope must be at the side of the oxygen cot to which the infant is returned. Intramuscular fluid is continued after operation. Providing no evidence of leakage such as a right pneumothorax develops, a few cc. of 5 per cent glucose may be given with a medicine dropper on the third day. If this is taken satisfactorily and the lungs remain clear regular small feeds of breast milk, also with a dropper, are begun. Should leakage occur, and a fluid level appear in the right pleural cavity, a water-sealed intercostal catheter is inserted at once, and gastrostomy performed. If the lung is kept well expanded the leak may heal. Continuity of the lumen must be demonstrated with a Lipiodol swallow before the gastrostomy can safely be abandoned. Some surgeons perform gastrostomy routinely about the third day to simplify feeding problems. Intravenous 4 per cent dextrose in N/5 saline may be required by dehydrated infants, but its use calls for great caution and no more than 75 cc. per kilogram per day should be given. Antibiotic therapy is continued during the early postoperative phase.

PHARYNGEAL POUCHES AND DIVERTICULA

MUCH the commonest type of oesophageal pouch is that in which the mucosa of the posterior wall becomes herniated through the weak point between circular and oblique fibres of the inferior constrictor of the pharynx (Fig. 36). As this triangular gap in the decussation is normally present some additional explanation for the formation of pouches must be sought and a clue to this lies in the fact that most patients are elderly men who give a long vague history



FIG 36

The pharyngeal mucosa is protruded between the decussating fibres of the inferior constrictor muscle to form a pouch.

of dysphagia predating symptoms due to the pouch. It seems probable that in these patients a chronic disorder of oesophageal peristalsis (chiefly marked by spasm of the cricopharyngeus muscle) exists, causing delay in swallowing, and eventually forcing the mucosa to bulge out through the only weak point. As the process continues food begins to lodge in the mucosal pocket, helping to distend it. Eventually this forms a true pouch which, as the weight of its contents increases, is slowly dragged down until it lies behind the gullet and pushes it forward. The more the latter is displaced the more is its

normal lumen occluded, and the distended mouth of the pouch instead comes into direct line with the pharynx and so tends to catch all food coming down until it is full, increasing still further its size and causing serious dysphagia (X-ray 119). Some patients find they can empty the pouch by pressing on the neck or twisting the head. The famous 'hanging judge', Judge Jeffries (whose temper may well have been soured by his affliction), suffered from such a pouch, and used to empty it periodically with a long-handled silver spoon.

The food in the pouch stagnates making the breath offensive, and gives rise to noisy gurglings as it is churned with air and saliva. Food particles spill from it, especially when the patient lies down at night, and may be inhaled into the lung to cause low-grade pneumonia. Some patients are able to sleep only when propped up in bed.

A barium swallow confirms the presence of the pouch, which must be distinguished from post-cricoid carcinoma as a cause of high dysphagia in elderly men. Oesophagoscopy calls for particular care as the beak of the instrument tends to enter the neck of the pouch, as does food, and may easily rupture it. If cricopharyngeal spasm is present it is overcome by the passage of bougies. If the pouch is large enough to justify surgery it is first washed out through a soft rubber catheter and penicillin solution instilled into the cavity. Parenteral antibiotics are also given.

Operation

The patient is placed in position as for thyroidectomy and an approach made either by a collar incision or by an incision along the anterior border of the left sternomastoid muscle. The left lobe of the thyroid is exposed and the middle thyroid veins and inferior thyroid artery tied and cut. The lobe is retracted medially, the carotid sheath laterally, to expose the pharyngeal wall. The fundus of the pouch is easily found if a rubber tube be passed into it, and is readily freed from the surrounding tissues for it is not usually adherent to them. As this pouch consists entirely of mucous membrane it must be handled delicately and never touched with toothed dissecting forceps. When its neck is defined it is divided, the tube having been first withdrawn, and the cuff sewn up with fine atraumatic interrupted sutures. Clamps are not used. The pharyngeal muscle layers are sewn together over the suture line, and the wound closed, a small tube draining the site. Fluids are allowed after 12 hours, soft solids in a week. Antibiotic cover is continued for a week.

Diverticula or pouches of the thoracic oesophagus are rare and symptoms caused by them rarer still. Most are discovered during

barium examinations for some other reason. They occur mostly in the lower third of the oesophagus and may either be due to tuberculous lymph glands becoming adherent to the oesophageal wall and drawing a portion of it out as a diverticulum, or to pulsion in similar fashion to a pharyngeal pouch. Very occasionally they cause pain or dysphagia. If big enough they may call for surgical removal similar in principle to that described for pharyngeal pouches. The possibility of a carcinoma lying in the oesophagus beyond the diverticulum must be kept in mind.

SIMPLE STRICTURES OF THE OESOPHAGUS

NON-MALIGNANT strictures of the oesophagus are rarely congenital, and most of those seen in small children are probably the result of acid regurgitation. The great majority are acquired in later life in the same way, and are therefore found at the lower end of the oesophagus often associated with an hiatal hernia needing surgical correction (X-ray 118). Their treatment is discussed in the section dealing with hernias (see p. 238). Other simple strictures follow the swallowing of corrosives or scalding water, or occasionally prolonged impaction of a foreign body, or operations upon the oesophagus. Like simple structures elsewhere in the body they respond to, and are best treated by, periodic dilatation and tend to recur, for 'once a stricture, always a stricture'.

The pharyngeal muscles open widely during the act of deglutition so that when caustic soda, lye, or strong acids are swallowed, or a child drinks from the spout of a boiling kettle, the resultant stricture tends to be well down the oesophagus, below the level of the aortic arch. In those who survive, stricture formation is indicated by slowly increasing dysphagia; although in some patients obstruction is sudden, due to solid food becoming impacted in the narrowed part. The nature of the lesion and its extent are determined by a barium swallow followed by oesophagoscopy. In contrast with what happens in cardiospasm, the oesophagus never becomes markedly dilated as a result of fibrous stricture. a channel, even if very narrow, remains, is lined by mucosa, and can be dilated.

Dilatation is initially attempted during oesophagoscopy. A fine gum-elastic bougie is first threaded through, followed by larger sizes. Further dilatations are carried out by sufficiently co-operative patients themselves. It is essential that they understand the process and that their confidence is won: refractory patients never do well. The advantages of self-bouginage are that it can be done as often as necessary, calls for no anaesthetic, and is helped by the swallowing mechanism (which is in abeyance during oesophagoscopy). The patient sits up, takes some deep breaths, and passes a fairly fine gum-elastic bougie, softened in warm water and lubricated with olive oil, into the mouth and begins to swallow it as soon as it reaches the pharynx. The lips are closed round it and its passage helped mainly

by continued swallowing but also by gentle manipulation. Force must never be used, and the patient never hurt. At the beginning only fine bougies can be swallowed but the size is gradually increased, and the largest bougie passed should be left in position as long as possible. This is at first done three or four times a day but later the intervals are lengthened. Solid food is encouraged as each bolus acts itself as a dilator.

This method usually succeeds. If it does not, or if ulceration is present, physiological rest is called for and therefore a gastrostomy is performed. After a time dilatation may prove quite easy. In more difficult cases the retrograde method should be tried: a ureteric catheter or even a thread, is swallowed and the end recovered through the gastrostomy. To it is attached a bougie with an eye in the tip and this is drawn back up the oesophagus guided by the thread or catheter until it passes the stricture. This is easier than passage from above because the lumen of the oesophagus below the stricture is a narrow cone leading to the orifice, whereas above the stricture it may be wide and irregular. Once a way through is established, bougies can be swallowed as before and the gastrostomy allowed to close.

Surgery is reserved for those strictures which resist dilatation or for those patients who refuse to dilate them. If the stricture is confined to a very narrow segment of the oesophagus it may be possible to excise it locally though this is the very type most suitable for dilatation. Otherwise the best operation is some form of intrathoracic oesophago-gastrostomy as performed for carcinoma, with high anastomosis in the right chest.

Strictures of 1 cm. or less in extent, especially those associated with hiatal hernia, may sometimes be successfully treated by extra-mucosal excision. The muscle coats of the oesophagus are incised above and below the stricture and the intact mucosal tube mobilized. The scar tissue constituting the stricture is then apparent, is seen to be largely extra-mucosal and is meticulously removed by sharp dissection, the mucosa itself being left intact.

INNOCENT TUMOURS OF THE OESOPHAGUS

INNOCENT tumours of the oesophagus are rare, though not so rare as once was thought, and over 70 per cent of them are leiomyomas, i.e. tumours of the smooth muscle coat. *Growths of the mucosal or submucosal layers project into the lumen as they grow and are therefore drawn farther and farther from the wall of the oesophagus by peristaltic action until their attachment to it becomes attenuated into a pedicle which sometimes allows spectacular regurgitation of the tumour into the patient's mouth.* Such tumours arise mostly just below the level of the cricoid or in the lower third of the oesophagus and comprise polypi, papillomas, adenomas, fibromas, epithelial cysts, lipomas and some leiomyomas. As they obstruct the lumen they are most likely to cause dysphagia as a presenting symptom; or they may ulcerate and bleed.

Intramural benign tumours on the other hand rarely cause dysphagia, for unlike carcinomas they do not give rise to a stricture of the lumen, even when they almost completely encircle it. The mucous membrane over them often ulcerates, however, giving rise to obscure but sometimes quite massive haemorrhages into the bowel. As their bulk increases, they may press upon an adjacent bronchus and cause collapse of a lower lobe, or a pleural effusion. The great majority of these growths are leiomyomas arising in the lower third of the oesophagus and extending in the plane between circular and longitudinal muscle coats. Occasionally they appear multicentric or diffuse in form, involving large segments of the oesophagus, but characteristically they are circumscribed even when large. Diffuseness must arouse suspicion of leiomyosarcoma. Of 9 such intramural growths described by Harrington in 1949, 7 were leiomyomas and 2 leiomyosarcomas. He suggested that innocent growths did not cause mucosal ulceration and that bleeding was therefore evidence of malignancy, but this is certainly incorrect.

Radiologically a leiomyoma may be seen as a lobulated opacity in the posterior mediastinum, moving on deglutition. During a barium swallow the mucosal folds are flattened over it, those opposite to it being normal; and the flow of barium past the tumour is unimpeded because the other wall dilates. Sometimes a distinct shelf is formed where the normal oesophageal wall meets the rigid segment

involved by growth. Oesophagoscopy is usually negative, the instrument passing the growth effortlessly, as does swallowed barium and as it is mobile it is not felt with the oesophagoscope. If, on the other hand, scars of previous ulceration are seen on the mucosa covering it, it is important in view of the surgical measures indicated *not* to perform a biopsy and so impair the integrity of the mucosa.

The treatment of choice is enucleation of the leiomyoma. As the growth is encapsulated this presents no technical difficulty providing the correct diagnosis has been made. The principal error to be avoided is unnecessary resection of the oesophagus. Every precaution is taken not to breach the mucosa during enucleation. To facilitate this a stomach tube is passed which can readily be felt in the mucosal canal, and provides a valuable guide to dissection.

A man of 45 suffered from intermittent attacks of melaena and haematemesis for two years. He had no indigestion and the first attack was preceded by pain in the chest. On admission his haemoglobin was 26 per cent. Recovery was rapid, but during it he developed pain in his left chest accompanied by pleural rub and followed by effusion. X rays showed collapse of the lower lobe of his left lung. Occult blood was sometimes present in the stools. Barium meals showed no signs of gastric or duodenal disorder, but a rounded opacity was noticed behind the heart shadow, and a mucosal shelf identified on barium swallow the barium flowing round a tumour. Oesophagoscopy was negative except for a scar in the mucosa of the posterior wall in the lower third of the gullet. It seemed probable that the tumour was a leiomyoma.

On opening his right chest I found a massive lobulated leiomyoma deep to the longitudinal muscles of the lower third of the oesophagus. It felt quite unlike a carcinoma, being curiously mobile, rather like a bag full of knuckle-bones. The oesophagus was mobilized the longitudinal muscle coat incised, and a white encapsulated tumour almost completely encircling the lumen shelled out. The leiomyoma was 15 cm. long and 5 cm. broad and the patient has had no symptoms since its removal.

CARDIOSPASM

A SEA captain of 40 complained that he had suffered for years from difficulty in swallowing solid food, although fluids and semi-solids caused no trouble. This hardly bothered him at ordinary times, but when he was on the bridge in foggy weather it was much worse. Food, he said, went down all right but then stuck. If he waited, or took a drink, it would sometimes pass, but he noticed that when he was particularly tired it did not, and after an increasing sense of fullness and discomfort he would have to leave the bridge, retch, and bring his food up again quite undigested.

An old lady of 72 said that she had had trouble with her swallowing for more than 30 years. It had increased in severity until she began to lose weight, and eventually sought medical advice fearing that she had a cancer of her gullet. She was taught to swallow mercury-weighted bougies, and this she had done at almost every meal for the past 15 years. During this time she had been unable ever to eat in a restaurant or dine with friends because of the inconvenience involved and the embarrassment it caused her. Now she wished to visit her daughter in Australia, and felt she could not continue to pass the bougies on the ship going out.

A man of 50 was referred as a case of chronic bronchiectasis. For several years he had had a cough productive of yellow sputum; and some time previously had had a small haemoptysis after which he was bronchoscoped at another hospital to exclude bronchial carcinoma. X-rays of his chest showed patchy mottling in the apical segment of the lower lobe of his right lung; and appearances which suggested a tumour bulging out of his superior mediastinum on the right side. On direct questioning he confessed that he was sometimes bothered by his swallowing and felt 'blown-out', but said that his cough was what he had come to see me about. Yes, his wife was always complaining about his bad breath.

These three patients suffered from cardiospasm. It was the cause of all their symptoms, and all were completely and permanently relieved by surgery. The old lady who had not swallowed unaided for 30 years, was able to do so without difficulty two days after her operation. The last patient required further treatment for the suppurative pneumonitis due to inhaled food fragments in his right lung.

Aetiology

The disorder which accounts for some 20 per cent. of cases of intrinsic dysphagia, is not due to failure of peristalsis, for in the earlier stages this is normal or even hyperactive though with continued obstruction to the passage of food passive distension occurs, and all contractility is lost. The oesophagus may assume vast proportions (X ray 120) taking up an S shape in the mediastinum so that it lies to the left inferiorly but bulges to the right in the superior mediastinum, where it may greatly resemble radiologically a mediastinal tumour. The aetiology of the condition, apart from doubtful evidence of its association with emotional disturbance, is quite unknown but there would seem to be an idiopathic spasm of the circular muscle fibres at the lower end of the oesophagus. As a rule no macroscopic change of any kind is visible. There are no adhesions, no inflammation no hypertrophy and no resemblance to congenital pyloric stenosis. The last few centimetres of the oesophagus are narrowed and blanched while above them balloons the thickened wall of the distended portion. The degeneration of the cells of Auerbach's plexus seen histologically is probably a secondary phenomenon occurring late in the disease, and not, as has been suggested, the cause of the disorder. As in any other trouble symptoms tend to be aggravated by worry or fatigue, as in the case of the sea captain but in general patients who suffer from it seem otherwise well adjusted, as was he. Far from finding much evidence of psychoneurosis among my patients, I have often marvelled at the equanimity with which most of them bore their inconvenient and distressing affliction. Nor is there the slightest evidence to suggest that psychotherapy is of any value.

The onset is usually insidious generally coming on in early adult life (though occasionally in childhood) and affecting rather more women than men but it may begin abruptly. It is in the latter case that patients relate it to some sudden shock or strain.

Symptoms

At first only solid foods seem to stick behind the lower end of the sternum, but after a pause, or drinking, pass through. At this stage there is often spasmodic pain, but this diminishes in frequency as time goes on and as the disease progresses pain is wholly absent. Periods of remission alternate with bouts of dysphagia, but gradually the condition grows worse until semi-solids and even fluids are arrested and may be regurgitated unchanged. Drinking usually helps food to pass, largely because when a sufficient 'head' or weight, accumulates in the oesophagus the constriction relaxes and lets the

contents flow through into the stomach. Patients learn various tricks to facilitate this, such as gulping air, drinking from a tap, or adopting odd postures. If help is not sought, nutrition is seriously impaired and vitamin B deficiency may appear. The patient becomes thin, wasted, and eventually half-starved.

As the distended oesophagus contains considerable quantities of undigested food, fragments spill out of it during sleep and are aspirated into the dependent lung where they give rise to chronic sepsis with cough, purulent sputum, and low-grade fever. The commonest lesion is chronic suppurative pneumonitis, but segmental or even lobar collapse due to bronchial obstruction, bronchiectasis or an acute aspiration abscess may ensue. Not infrequently the patient seeks advice for these manifestations rather than for his dysphagia, with which he has learnt to live. In addition the food accumulation may set up oesophagitis with ulceration of the mucosa.

Investigations

Plain radiography of the chest is followed by a barium swallow. The former shows whether or not a lung lesion is present calling for preliminary treatment with antibiotics, postural drainage or bronchoscopy. The barium swallow shows arrest of the barium at the cardia, a thin trickle or none at all reaching the stomach. The oesophagus above is distended—usually to a degree far greater than occurs with a malignant stricture—and is smooth-walled and flask-like. Its size and shape depend upon the length of the illness, but the lower end is always rounded and symmetrical, with none of the irregularity usually seen when a growth is present. In well-established cases no peristalsis is visible. If amyl nitrite is inhaled relaxation at the sphincter occurs and barium flows through into the stomach, as of course it cannot do in the presence of an organic stricture.

In spite of these clear distinctions, oesophagoscopy is still necessary to exclude quite finally the possibility of carcinoma, peptic ulceration, or reflux oesophagitis being the real cause of the stenosis and dysphagia. Before this is carried out (and certainly before an anaesthetic is given) the oesophagus must be washed out lest food and fluid flood back into the pharynx and drown the patient.

Treatment

The treatment of cardiospasm by the passage of mercury bougies is wholly outmoded and there is no place for them in modern management. Their use condemns the patient to a lifetime of intolerable discomfort and inconvenience and drastically curtails social life as he

can never dine out with friends, travel, or visit a restaurant. Some patients indeed almost live the life of hermits.

If the patient is seen soon after the onset of symptoms, the best procedure is dilatation of the contracted segment with a *Negus hydrostatic bag*. After treatment with antibiotics has eliminated any pulmonary sepsis present, and the oesophagus has been cleaned by daily washouts with dilute sodium-bicarbonate solution, a general anaesthetic is given and the oesophagoscope passed. The constricted area is inspected and a wide gum-elastic bougie passed through into the stomach. After this has been withdrawn it is followed by the hydrostatic bag on an introducer. When the bag is snugly gripped by the oesophageal muscle it is slowly filled with water so that the muscle fibres are fully and evenly stretched. In many cases this will afford complete and permanent relief but if it should not do so, or if the disorder is severe or long-established, it is a mistake to discourage a possibly already despondent patient, or to waste time in repeated dilatations. *Heller's operation* should be proceeded with at once.

If lung infection is present it must of course be dealt with before operation and the oesophagus washed out as before. A high calory vitamin-fortified diet, avoiding solids, is given but the most effective way to improve nutrition is to relieve the obstruction, and if this is done the patient's general condition improves far more rapidly than by any other means.

Heller's operation

The best approach to the affected part is through a left postero-lateral intercostal incision in the seventh space. The lower lobe of the lung is packed upwards under warm moist towels and the layers of the inferior pulmonary ligament opened. The lower end of the oesophagus is mobilized mostly by blunt dissection, and care is taken not to open the opposite pleura. A plastic tape is passed round, and the oesophageal hiatus cleaned of fat and areolar tissue, so that the cardia can be drawn up to expose the upper two inches or so of the fundus near it. It is usually quite easy to do this, but if any difficulty is experienced in getting good exposure a short incision in the diaphragmatic muscle is made and afterwards repaired.

The oesophagus is lifted firmly with the fingers of the left hand and the longitudinal and circular muscle coats cut with a knife until the mucosa appears and bulges up in the incision (Fig. 37). Great care must be taken not to injure it but if an accidental nick should occur, and the lumen be opened, it is at once repaired with a few

fine interrupted sutures. Real danger lies in a wound occurring and passing unnoticed. The incision through the muscles is carried up the oesophagus for about 3 inches and down on to the fundus for 2 inches more. Failures are due to this myotomy being insufficient in length. Bleeding is very slight, only the vascular ring round the cardia

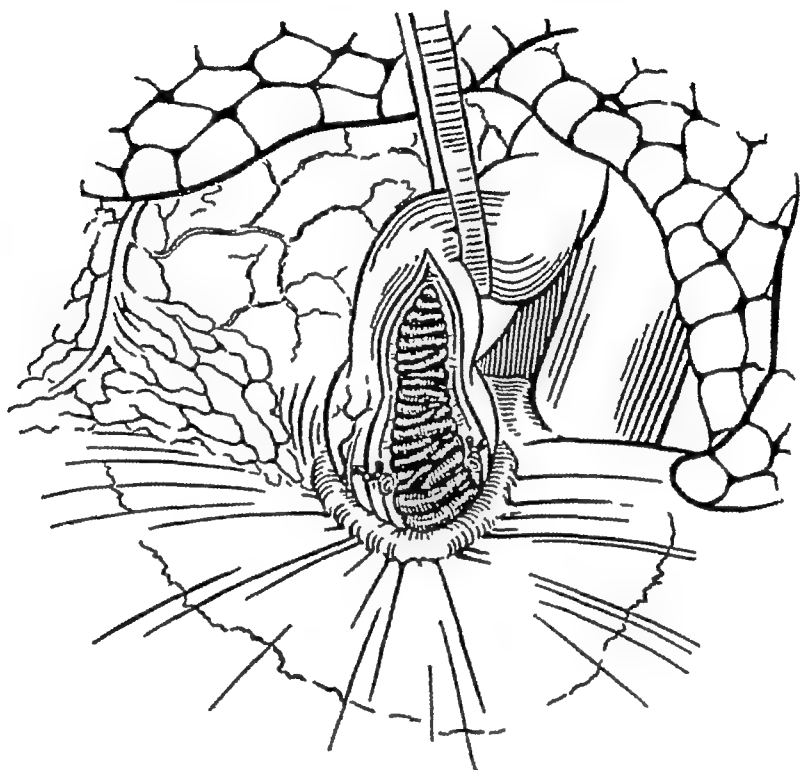


FIG 37. *Heller's operation*

The muscle coats of the lower end of the oesophagus and the stomach wall have been incised and the mucosa bulges through

or an occasional small vessel requiring ligation. The mucosa is examined to make sure no strands of muscle over it have been overlooked, any found being lifted and cut with scissors. Nothing is sutured, and the chest is closed without drainage. There is no need to leave a tube down into the stomach, and after 24 hours of fluids, meals of minces and purees are begun followed by normal diet in three or four days. Patients get out of bed within 48 hours and leave hospital at the end of ten days. The results of surgery are almost uniformly excellent and the condition does not recur. Operations such as oesophago-gastrostomy or cardioplasty are wholly unnecessary, dangerous, and in every way contra-indicated.

CANCER OF THE OESOPHAGUS

THERE is probably no malignant disease which causes greater misery than cancer of the oesophagus and it may truly be said of it that surgery holds out a poor hope of cure, but a good hope of palliation. Although cure should be the surgeon's aim, palliation is well worth while. Treatment, such as gastrostomy, which does not achieve this is better not done at all.

Let us consider the results of some of those with most experience in resecting growths of the oesophagus in 1952 Sweet reported exploring 182 patients with growths in the mid oesophagus, resecting 120 of them, with 30 deaths, and a 4 per cent. 5-year survival, in 242 growths of the lower oesophagus 167 resections were carried out, with 20 deaths, and 17 per cent. were alive 5 years later Garlock in 1954 reviewed 457 cases of which 451 were explored, but in only 180 was a resection performed Fifty seven of these died postoperatively and 20 survived 5 years By 1955 Clagett had seen 475 patients at the Mayo Clinic with cancer of the lower oesophagus, rejected 219 of these as inoperable, exploring 256 but resecting only 131 Twenty two died postoperatively and of the 45 who had been operated upon for more than 5 years 11 survived.

There are the results of experts, and they may be thought to present a gloomy picture, but of those patients of Clagett's who were *inoperable* 88 per cent. were dead in a year and 97 per cent. in eighteen months Of Sweet's 90 who survived mid-oesophageal resection 58 per cent. were alive a year later and of those with lower-end resections, 87 per cent. Sixty-three per cent. were living after 2 years Furthermore all these as well as those dying earlier could at least swallow their food in comfort as long as they lived No one who has seen a victim of cancer of the gullet dying of starvation, saliva he cannot swallow dribbling over his chin, mental distress almost equalling physical disease will be inclined to underestimate the boon of palliation. *To such relief gastrostomy contributes nothing* half the patients so treated never leave hospital. The average time of survival after gastrostomy is 120 days.

Incidence

Like cancer of the lung, cancer of the oesophagus is 8 or 9 times

more frequent in men than in women (in whom it is usually post-cricoid, and follows the Plummer-Vinson syndrome of hypochromic anaemia and high dysphagia) It is much less common than bronchial carcinoma, however, comprising only 5 per cent of cancers and accounting for some 2,000 deaths a year in Great Britain. The low rate of operability and the disappointing results of surgery are not due to greater malignancy in this than in other growths, but simply to the fact that cancer of the oesophagus does not cause symptoms until too late. Otherwise, it resembles comparable growths elsewhere in the body. In over 1,000 autopsies Ochsner found in 418 (41 per cent) no evidence of metastatic spread at all. Many patients die of starvation, not of extension. As the oesophagus is lined by squamous epithelium most growths are squamous-celled; but at the lower end many adenocarcinomas occur, either growing upwards from the stomach or arising in ectopic gastric mucous membrane. All are prone to extend up or down the oesophagus in the submucous layer, so that a substantial cuff of apparently healthy tissue must always be left on both sides of a resected neoplasm. It is convenient to divide them into those growing in the upper third of the oesophagus (from pharynx to aortic arch); those in the middle third (from aorta to inferior pulmonary vein); and those of the lower end and cardia. In a series of 8,572 cases the incidence in these regions was, upper third 20 per cent; middle third 37·2 per cent.; and lower third 42·8 per cent. (of which no doubt a proportion should be regarded as growths of the stomach) Upper third growths metastasize to mediastinal and supraclavicular glands; those in the middle third to the carinal glands; but 30 per cent of middle third, and 50 per cent. of growths at the lower end, metastasize directly below the diaphragm. Any operation, therefore, designed to extirpate, must aim at the removal of the glands along the lesser curve of the stomach and the left gastric artery.

Symptoms

Difficulty in swallowing is usually the first, and often the only symptom. Cancer of the oesophagus is responsible for 80 per cent. of cases of intrinsic dysphagia. Its onset may be sudden, due to muscular spasm rather than blockage by growth, so that later swallowing improves for a time before malignant stenosis supervenes. Usually the onset is gradual, the patient noticing, long before frank dysphagia, that he is taking longer to eat his meal, or that he is not completing it; and he may describe his vague discomfort as 'indigestion'. Nor is a long history of dysphagia always grounds for com-

placency for a growth occasionally develops in relation to an hiatal hernia, or to cardiospasm, or as a rare termination to the Plummer-Vinson syndrome. Sometimes the first symptom is a purposeless cough, coming the patient hardly knows whence but in fact arising from a sense of oesophageal discomfort. There may also be vague malaise and ill health, hiccup due to phrenic irritation, or fever caused by infection in a necrotic mass. As the cancer grows, difficulty is experienced in swallowing dry solids, then comes occasional regurgitation then dysphagia with moist foods, and later with semi solids. The level at which food seems to stick can often be localized with fair accuracy. Weight is lost, not so much through the wasting of malignancy as from inanition, and the patient may present himself in a state of semi-starvation. Pain between the scapulae, boring in character, indicates invasion of surrounding tissues and an irremovable growth. Hoarseness, as with cancer of the lung, is a late and grave sign of recurrent laryngeal nerve involvement. Careful distinction should be made between loss of appetite and loss of ability to eat, the former suggesting dissemination.

Investigation

First the patient is asked to eat and drink so that his difficulties can be directly observed. During physical examination particular attention is paid to the palpation of the liver for metastases and the left subcostal region for the presence of a craggy primary tumour of the stomach. (For this the patient should lie on his right side.) The supraclavicular fossae are felt for enlarged glands, and a rectal examination must never be omitted, for it may reveal a shelf of peritoneal secondary implants. Plain radiography of the chest rarely gives any indication of the primary tumour but as the venous return from high gastric and oesophageal areas enters the inferior vena cava directly instead of passing through the portal system, secondary deposits in the lungs may be visible before there is any indication of them in the liver. The stools are examined for occult blood, and the degree of anaemia and derangement of plasma proteins estimated.

Conclusive information is usually gained by screening the patient while he swallows barium. Its passage is observed and delay, spasm, filling defect, or disorder of peristalsis noted. A malignant stricture is likely to appear craggy and irregular a tortuous track of barium winding its way through the lumen left by the growth. Furthermore, it may occur at any level, and not always at the point where the oesophagus traverses the diaphragm, as is the case in cardiospasm. Distension above the block is also much less and is not so smoothly

symmetrical Such differences are the rule, but dangerous exceptions occur, and the indirect evidence of radiography must always be verified by direct inspection Some malignant strictures look regular and innocent; whereas those due, for example, to peptic ulceration may appear malignant If the patient is placed in the Trendelenburg position the fundus of the stomach is outlined with barium and herniation or reflux is tested; should a stricture be present this manœuvre allows its lower margins to be examined.

Oesophagoscopy is imperative in all cases of dysphagia, and whenever there is suspicion of cancer of the oesophagus. In passing the instrument great care must be exercised because of the increased risk of perforation When the suspected growth or ulceration is encountered a biopsy is taken from it, an estimate made of its mobility, and its distance measured from the incisor teeth It must be remembered that a growth may lurk beyond an innocent stricture, or be hidden by inflammatory granulations, or by a pouting lip of oedematous mucosa

Finally, a bronchoscope must always be passed to exclude direct infiltration of the bronchi or trachea, or even erosion and fistula formation at the point where the oesophagus lies closely related to the left main bronchus; and also to make sure that a bronchial carcinoma is not the cause of dysphagia rather than an oesophageal growth

Preoperative care

Before operation every effort is made to restore nutrition, and combat dehydration. A two-hourly fluid or semi-fluid diet of at least 3,000 calories a day is begun, supplemented by ascorbic acid and other vitamins No solid food which may block the narrowed lumen is allowed under any circumstances, although a fluid diet tends to be both monotonous and unpalatable. Between feeds the patient is encouraged to drink whatever he wishes, and as frequently as he likes Only in those who are quite unable to swallow fluids should a gastrostomy be done, and then it should be of the simple inkpot, Stamm type. If anaemia is present a blood transfusion is given, and breathing exercises and antibiotic therapy are begun Oral sepsis, which is frequently present and may have some aetiological significance, is treated by scaling, by removal of loose teeth, and by mouth washes, but not by wholesale extractions If the patient is well enough he is encouraged to move about freely and keep out of bed In some, preliminary digitalization is wise.

Operation

The aim of surgery is to restore the ability to swallow. When the patient awakes from his anaesthetic it should be possible to tell him that this has been given back to him. If therefore, removal is out of the question a short circuit must be contrived. Multiple stage operations and antethoracic reconstructions are outmoded. Seldom completed, carrying in themselves an immediate mortality of over 30 per cent. and a much higher morbidity they have no part in modern surgery.

Save in exceptional circumstances hypopharyngeal and post cricoid growths (i.e. in the upper third of the oesophagus) demanding laryngectomy and complicated plastic procedures, are probably best treated by deep X ray therapy especially as they are readily accessible and not buried deep in the thorax.

When the middle and lower thirds of the oesophagus are involved, the treatment of choice is radical excision and restoration of continuity by oesophago-gastrostomy the stomach being first mobilized and then drawn as high into the thorax as may be necessary.

Growths in the lower third (i.e. below the level of the inferior pulmonary vein) are approached by a thoraco-abdominal incision running along the line of the left seventh rib which is excised and curving down over the costal margin to end as a vertical laparotomy above the umbilicus. The operability of the growth in the oesophagus is first determined, and a search made for secondary deposits on the lesser curve of the stomach or in the liver. Even if some are present, oesophago-gastrostomy is worth while. If there seems a chance of real extirpation it is necessary to perform a radical removal of stomach, spleen, tail of pancreas and the related lymphatic field. It is in any case a convenience to remove the spleen when mobilizing the stomach to avoid troublesome bleeding from it and the dissection of the short vessels between the two. The stomach is mobilized by dividing the gastro-colic omentum along most of the greater curvature, followed by the lesser omentum. The gastro-epiploic vessels are preserved and the left gastric artery identified, and cut between ligatures.

The oesophagus lies in the mediastinum surrounded by loose areolar tissue and its mobilization is largely achieved by blunt, digital dissection, with ligature and division of the vessels as they are encountered, care being taken not to open the opposite pleura, or to repair tears in it as soon as they are made. The necessity for sharp

oesophagus is mobilized and divided above the neoplasm, its lower end closed, and an anastomosis with the fundus of the stomach carried out in similar fashion, except that no resection is performed and the lower oesophageal segment and the stomach remain in continuity.

Postoperative care

Postoperatively the drainage tube is removed after 24 or 48 hours when radiographs show the lung to be fully re-expanded, and subsequent effusions or air are aspirated. During the first 48 hours blood replacement (comprising usually 3 or 4 pints) is completed; and followed by an intravenous drip of about 3 litres of 4 per cent. dextrose in N/5 saline a day, to which is added 250 mg of ascorbic acid, 150 mg. of nicotinic acid, and 5 mg of riboflavin. The quantity of intravenous fluids given depends on the blood chemistry and clinical state of the patient. His mouth is regularly cleaned with glycerine and water and he is given penicillin lozenges to suck. Nothing is swallowed before the third day, when sips of water are begun providing no ileus is present. On the next, 1 oz. of glucose fruit drinks is allowed hourly; and on the fifth and sixth days, 2-oz hourly feeds of peptonized milk with glucose and egg, rising to 4 oz by the end of the week. Amounts are increased thereafter, until by the tenth or eleventh day a more varied semi-solid diet is begun with greater intervals between feeds. On the fourteenth day solids are taken, and in another week a normal diet.

Throughout the early stages great attention is paid to full expansion and aeration of the lungs, and the patient is made to cough hourly when awake. Atelectasis and accumulating secretions call for prompt bronchoscopy under local anaesthesia in bed. The breathing and leg exercises taught before operation are carried out, and enough sedation is given during the first few days to prevent these, and coughing, from being too painful. Antibiotics are continued for about ten days. As many patients are elderly and frail, atrial fibrillation is not uncommon and must be controlled by prompt digitalization.

In the early postoperative phase paralytic ileus, signalled by distension and absent bowel sounds, must be watched for, and is an indication for further intravenous therapy, and continuous aspiration of stomach contents through a Ryle's tube, which some surgeons leave *in situ* at the end of the operation. Ileus may be mimicked by pylorospasm, which is apt to follow section of both vagi, and gives rise to distension, though auscultation confirms the presence of bowel sounds. This is often relieved by a 25-50-mg pastille of

Mecchothane taken sublingually after each feed later on the vagotomies may also cause troublesome diarrhoea. By far the most serious and usually fatal, complication of the operation is leakage from the anastomosis. This leads at once to the appearance of air and fluid in the pleural cavity and a marked deterioration in the state of the patient. Aspiration of the fluid reveals oesophageal contents. Empyema soon follows. When such a breakdown is not due to faulty stitching it is more likely to follow ischaemia of the stomach than of the oesophagus. If the accident is recognized early and the condition of the patient permits, the best hope of survival lies in reopening the chest and attempting to repair the fistula but if circumstances do not allow this the only alternative is to cease oral feeding, aspirate the stomach, insert a water-sealed drainage tube into the chest, and endeavour by continuous suction upon it to maintain full expansion in the lung and hope that the leak will heal. The outlook, however is usually poor.

PART THREE
SURGERY OF THE HEART AND
GREAT VESSELS

CARDIAC ARREST

If neither pulse nor apex beat can be felt cardiac arrest must be assumed. Urgent action is required and little time should be lost listening with a stethoscope much less obtaining electrocardiograms. The heart may stop beating during an operation or the induction of anaesthesia for one of two reasons

(1) in the great majority it stops simply because *not enough oxygenated blood is reaching the heart muscle through the coronary arteries*

(2) because *ventricular fibrillation occurs*, in which the myocardial bundles contract in a completely uncoordinated way so that no blood is expelled from the ventricles, which resemble a writhing bag of worms.

Of these the first is much the commoner, and (providing the coronary arteries themselves are healthy) may be due to mechanical obstruction of the airway at some point to inadequate aeration of the lungs, or an excess of anaesthesia causing respiratory paralysis, to a sudden drop in blood pressure (such as occurs during haemorrhage or during the act of valvotomy) robbing the coronaries of their supply or to direct interference with the heart action. The best treatment, it goes without saying, is prevention that is maintenance of good oxygenation and an adequate blood pressure throughout every operation, a responsibility shared by surgeon and anaesthetist. No measure of resuscitation is any use which does not restore oxygen to the myocardium. A tube must be passed into the trachea if one is not already there oxygen and a bag to squeeze it in with must be at hand and a knife available to open the chest. These and knowing what to do and doing it quickly are the only necessities of cardiac first aid.

When the heart stops it must be restarted within three minutes or irreparable damage to the brain occurs. It follows that considerations of surgical sterility are quite secondary. Warning signals of imminent danger during an operation must be recognized and respected. Among these are blueness, a poor blood pressure, slow feeble heart beats, and cardiac irregularities such as extra systoles paroxysmal tachycardia or bouts of atrial fibrillation also electrocardiographic evidence of anoxia, if continuous readings are being taken, such as

bundle branch block or S-T depression. Any of these are an indication for the lung to be reinflated with pure oxygen, and the heart to be rested

If the heart stops two things must be done:

(1) an efficient airway must be established, and respiration with oxygen begun;

(2) the chest must be opened, if it is not so already, and effective cardiac massage commenced. The abdominal trans-diaphragmatic route is not satisfactory, but if the abdomen is already open it can be used. No time is wasted on aseptic precautions, but a knife seized, and the thorax opened in one rapid incision through the fourth or fifth intercostal space. If the heart is motionless, in asystole, the ventricles are grasped and firmly—but not roughly—compressed and released rhythmically to ensure that blood is pumped into the coronaries and up the carotids to the brain. This can be done better if the pericardium has also been opened; and it should be continued so long as any hope of restarting the heart remains. Rapid transfusion, preferably into the aorta, is essential if the arrest is due to blood loss. Usually the heart recommences its beat after a short period of massage. Should it not do so massage is supplemented by injection into the atrium of calcium chloride 2 to 5 cc. of 10 per cent solution; followed if need be with 0.5 cc. of epinephrine 1:1000 in 10 cc. of isotonic saline. Any normal heart can be made to beat again if the right things are done soon enough, and there is no excuse for not trying

Once the heart is visible it is important to diagnose for which of the two reasons, anoxia, or ventricular fibrillation, it has stopped, as their treatment differs. The treatment of anoxia, much the commoner, has been dealt with; and if ventricular fibrillation is present massage is still vital to maintain a coronary blood flow, and may succeed in restoring normal rhythm. However, it may be necessary to restart normal beating by temporarily causing abrupt arrest of all the muscle twitchings by passing an electric shock through the heart with a defibrillator and subsequently restoring normal contractions by massage. Two electrodes—rather like sugar tongs and covered with moist sponge—are placed one on either side of the ventricles, and a shock of 110–130 volts for 0.1 second passed through. This is usually sufficient, but if it fails and fibrillation starts again, the voltage is raised by steps of 30–40 volts, and the process repeated up to 200 volts if necessary. Between each shock massage is continued. Ventricular fibrillation is most apt to occur during manipulation of congenitally abnormal hearts, and one of the particular

dangers of hypothermia is the greatly increased risk of fibrillation at temperatures below 28° C. This may occur while the temperature is being lowered before the operation has even begun. Above 28° C. the risks are much less and in practice patients are not now cooled below 30° C. Should ventricular fibrillation occur in the early phases of an operation (i.e. before an atrial septal defect is closed, or a pulmonary stenosis relieved) the procedure should be completed as rapidly as possible before defibrillation is carried out.

ANGIOCARDIOGRAPHY AND CARDIAC CATHETERIZATION

ANGIOCARDIOGRAPHY

THE injection of opaque media into the circulation to delineate the heart chambers and great vessels on X-rays provides valuable information, but is by no means devoid of risk, carrying a direct mortality of 1-2 per cent. It is chiefly of use in complicated congenital heart lesions in which the diagnosis remains obscure after the usual clinical, radiographic and electrocardiographic investigations. It consists in rapidly injecting a quantity of dye which traverses the heart as it were in a single package, without becoming diffused as it would do if it were injected slowly. Serial X-rays are taken of its progress so that pictures are obtained of all the heart chambers and their out-flow tracts (X-ray 121).

If a 70 per cent solution of diodone (varying between 10 cc. in an infant to 60 cc in an adult) is injected into an arm vein it reaches the right atrium in 0.5 second and passes through the heart and out into the aorta in about 5 seconds. In recent years it has been seen that great advantages lie in combining cardiac catheterization (by which the haemodynamics of the heart can be studied) with angiography (which depicts the anatomy) in a single operation, especially as this permits dye to be injected directly through the catheter into any chamber which it is particularly desired in outline. Serial radiographs at exposure frequencies of up to $\frac{1}{2}$ second are taken in two planes at right angles to one another on a continuously moving roll of X-ray film so that three-dimensional study is possible, and repeated injections of dye, in which the chief risk of angiography seems to lie, are avoided.

Urokon is preferred by many to diodone as having less toxicity; not more than 1.2 cc per kilogram of body should be employed, and repeated examinations on the same day are absolutely contra-indicated.

CARDIAC CATHETERIZATION

The technique of passing a catheter into a suitable arm vein and thence on into the right side of the heart provides three valuable sources of information. (1) The catheter itself is radio-opaque so

that its course within the heart and great vessels can be watched and the catheter manipulated as though it were an exploring probe by which cardiac malformations may be detected. For example, it may be possible to pass the catheter from right to left atrium through a patent foramen ovale or atrial septal defect. (2) Pressures within the heart chambers or great vessels are measured by connecting the catheter with a saline or electrical manometer. In this way the presence and exact location of a stenosis is determined and the onset of such conditions as pulmonary hypertension detected before being clinically manifest. (3) Blood samples can be taken from within the heart and their oxygen content analyzed. This provides useful information about the position and size of abnormal shunts of blood from one side of the heart to the other (X ray 127)

HYPOTHERMIA

If the body temperature is lowered cellular metabolism is slowed down and oxygen requirements of the cell are correspondingly reduced. For example, at 31°C oxygen demand is roughly 55 per cent. of normal. In addition enzyme activity is depressed, and it is this activity that normally leads to damage or death of the cell in anoxia. This, briefly, is the physiological basis for the use of hypothermia in surgery; and it can be achieved practically in a variety of ways by cooling the whole skin surface of an anaesthetized patient with ice-water, or chilled blankets, or some similar means of refrigeration, by perfusing the peritoneal or pleural cavities with cold saline, or by cooling the blood stream directly. The first and the last are the methods employed in man. Cooling the body surface is simple to achieve and relatively safe providing temperatures below 28°C are not required. Both rectal and oesophageal temperature readings are taken continuously. The oesophageal temperature is a near approximation to that of the heart and brain, the rectal to that of more peripheral tissues. It must always be remembered that temperature continues to drop by 2 or 3 degrees after the ice packs are removed, and allowance is made for this lag. Direct cooling of the blood offers certain advantages. It is in itself a form of anaesthesia and therefore requires only minimal supplementation by conventional means, it avoids certain inherent dangers of the other methods, for example loss of platelets which promotes a tendency to bleed, and a drop in temperature when circulation begins again in surface-cooled subjects.

Hypothermia is by no means devoid of risks and drawbacks, and some problems associated with it still await satisfactory solution. Of these the chief is certainly the greatly increased irritability of the heart muscle at low temperatures, especially when handled, resulting in ventricular fibrillation and consequent cardiac arrest. This irritability seems due to loss of potassium from the cells, for cooling alters the blood pH so that water enters the cells and potassium leaves. It is unusual at temperatures above 28°C , but becomes a serious danger below this level. Treatment of fibrillation consists in passing an electric current through the heart muscle (see p. 280). To stop the circulation completely for five or six minutes a tem-

perature of 30°C is sufficient but to arrest it for longer, as is necessary for some forms of intracardiac repair the temperature must be lowered to the region of 25°C , when the problem of fibrillation becomes serious. Although this is the worst difficulty there are also a number of others due to disturbance of metabolism, and not all are clearly understood. It follows therefore that the period of hypothermia should be as short as possible and that the patient should be returned to a normal temperature as quickly as can be arranged.

In an attempt to overcome some of these problems Brock has evolved a direct method of refrigeration. Instead of cooling the patient preoperatively and thereby inducing a phase in which ventricular fibrillation might occur before the chest is open and the heart accessible for emergency treatment, he first performs thoracotomy at normal temperature, examines the heart and establishes the diagnosis. If hypothermia is required one catheter is inserted through the right atrial appendage into the superior vena cava, and another by the same route into the inferior vena cava. The blood thus withdrawn from the top catheter is pumped through a refrigerating coil and returned through the inferior tube the cooling process taking about half an hour. Speedy cooling is dangerous and should not exceed 1°C in four minutes for children or in six minutes for adults. When the operation is complete rapid re warming to the region of 35°C is secured by the same method. Both catheters lie disadvantageously in the limited operation field.

At the London Hospital we employ a neat surface-cooling unit incorporating refrigerator motor and defibrillator. Cooled glycol circulates in a number of rubber coils arranged on the anaesthetized patient, and re warming is by the same method. Others use immersion in an ice-water bath which can be wheeled into the theatre.

Hypothermia is required for direct attacks upon the pulmonary valve and infundibulum (p. 302) and in operations for closure of atrial septal defects (p. 310).

Procedures needing longer circulatory arrest than eight minutes (i.e. closure of ventricular septal defects) demand an artificial heart lung, or crossed circulation donor, and these techniques remain in an early stage of development.

CONGENITAL DEFECTS OF THE HEART AND GREAT VESSELS

THE evolution, during foetal life, of the simple pulsating tube of the primitive heart into the elaborate four-chambered organ of the adult, by a process of fusion and convolution, is complicated still further by the varying fates of the original paired aortas and their symmetrical branches to the branchial arches. Defects may occur because blood channels persist which normally disappear, leaving such structures as anomalous aortic trunks or a patent ductus arteriosus, or the process of obliteration goes too far, as seems to happen in coarctation of the aorta and pulmonary atresia, or the fusions of tissue which divide the S-shaped loop of the foetal heart into four chambers are incomplete, leaving atrial or ventricular septal defects; or a mixture of all these things may give rise to the multiple complexities of Fallot's tetralogy.

The results of the derangements of these strange processes of growth and ablation, the mysterious selection of one route and the rejection of another, are in practice remarkably consistent. Fallot's tetralogy, septal defects, pulmonary stenosis, patent ductus and coarctation together account for 80 per cent of their total. Their importance, however, should not be exaggerated when seen in the perspective of heart disease as a whole, for sufferers from congenital defects account for only 2 per cent. of all cardiac patients; and the problem posed by them is in no way comparable in magnitude to that of rheumatic or degenerative heart disease. They have for surgeons a particular interest because their treatment challenges ingenuity and skill, and represents the frontier upon which most spectacular advances have been made in recent years. In some, excellent and consistent results have already been won and operations are solidly established; in others difficulties remain, and their solution awaits further development of such techniques as hypothermia, crossed circulation, and artificial blood pumps making surgery under direct vision in the dry and open heart a safe and standard procedure.

On the whole the diagnosis of congenital heart disease does not call for elaborate investigations such as cardiac catheterization, blood-gas analyses, or angiocardiology (although these were essential in establishing and confirming characteristic findings in early

series and are still necessary in doubtful and complicated cases), but can usually be made on clinical and radiological grounds alone. Irrespective however of what indirect tests have been carried out before operation, or what theoretical conclusions reached, no substitute exists for the surgeon's direct examination with his eyes and fingers in the open chest and it is to this final court that cardiology owes most of its recent advances.

Congenital defects of the heart fall naturally into two groups—those with central cyanosis and those without. Central cyanosis is caused by unoxygenated venous blood from the right side of the heart mingling with arterial blood from the left side. Almost the only way in which this can happen in congenital heart disease is by means of

(1) a defect in the septum, sometimes between the atria or, more often, between the ventricles,

(2) some other factor, such as pulmonary stenosis, which obstructs the normal outflow from the right ventricle causing it to hypertrophy so that the pressure in it rises enough to drive venous blood from right to left through the septal defect, normally flow would be from left to right. This is exactly what is seen in Fallot's tetralogy. Sometimes obstruction is not in the pulmonary artery itself but more distally in the vascular bed of the lung arterioles and capillaries. Such *pulmonary hypertension* may come about as a result of exposure of these vessels to abnormal stress, so that in some long-standing cases of patent ductus arteriosus the flow in the ductus becomes reversed, and central cyanosis follows. A similar result is seen in Eisenmenger's complex where all the anomalies of Fallot's tetralogy exist save pulmonary stenosis. There is often very severe pulmonary hypertension in mitral stenosis with right ventricular hypertrophy but no central cyanosis occurs because there is no septal defect although *peripheral cyanosis* is a feature of the disease. On the other hand the mere existence of a ventricular septal defect may eventually produce a reversal of the blood flow through it, for as time goes by the right ventricle hypertrophies in response to the pressure from the left until it exceeds it, and drives the blood back causing cyanosis.

It is most important to distinguish *central cyanosis* from the *peripheral cyanosis* which everyone suffers in cold weather, and which is also seen in patients whose hearts are failing, or whose cardiac output is diminished for any other reason, such as mitral stenosis or constrictive pericarditis. This is readily done by the following features

(1) Central cyanosis is present on surfaces which are themselves

warm, such as the buccal mucous membranes, the tongue and the conjunctivae.

(2) It is nearly always associated with polycythaemia and an increased haemoglobin level, although the haemoglobin is not raised in proportion to the cells

(3) There is clubbing of the fingers and toes.

(4) It is usually obvious at rest in a warm environment, becomes worse on exertion, and is then accompanied by marked dyspnoea.

Peripheral cyanosis on the other hand is the kind with which we are all familiar, resulting in blue noses, ears and finger-tips, and blueness of the outside of the lip rather than the inside. It is never associated with clubbing or polycythaemia, unless of course there is some other cause for them.

Fallot's tetralogy accounts for 60 per cent of the defects in which central cyanosis is a feature. Others are pulmonary or tricuspid atresia, both of which closely resemble it, but in which the prospects of successful surgery are not so good, transposition of the great vessels; Eisenmenger's complex; and a few other rarer and complicated anomalies. Simple pulmonary stenosis does not cause cyanosis unless an atrial defect, or patent foramen ovale, is present through which blood can be shunted when pressure in the right heart rises sufficiently. The lesion thus falls sometimes in one group, sometimes in the other.

The anomalies which are *not* associated with cyanosis are those in which either the shunt is from left to right or none at all is present, and the lesion is an obstructive one. Among those with left to right shunts are patent ductus arteriosus and atrial and ventricular septal defects; and obstructive lesions include coarctation of the aorta, aortic stenosis, and simple pulmonary stenosis without a patent foramen.

Once the cyanotic have been distinguished from the non-cyanotic group, the question must be asked. *'Is blood flow through the lungs more or less than usual?'* Cyanotic patients who have a *decreased* flow (as occurs in Fallot's tetralogy and pulmonary stenosis with interatrial shunt) can usually be helped by surgery; but those in whom it is *increased* (as in transposition of the great vessels and some types of tricuspid atresia) cannot. In the acyanotic group pulmonary blood flow is *increased* in patent ductus arteriosus, and atrial and ventricular septal defects, the first two of which are readily operable, the other now entering the orbit of surgery, but it is *decreased* in simple pulmonary stenosis.

Assessment of this flow depends upon radioscopic appearances.

If pulsation can be clearly seen in the main pulmonary vessels, and their shadows are prominent if the hila seem congested and heavily striated and if patchy shadows are visible over the lung fields, it is safe to assume that the volume of blood reaching the lungs is increased. If little or no pulsation can be distinguished at the hila, even if the main pulmonary trunks seem to be present, and if the lung fields themselves are unusually translucent and lacking in peripheral markings pulmonary ischaemia is present and surgery will probably benefit the patient. In cases where doubt remains cardiac catheterization or angiography may be necessary to settle it.

The radial pulse is small or normal in all forms of congenital cardiac defects except in patent ductus arteriosus, when it is of the Corrigan collapsing type and in coarctation of the aorta, when it is hypertensive, this being also the only congenital condition in which the systemic blood pressure in the arms is raised.

The apex beat helps to distinguish between left and right ventricular hypertrophy the former resembling the blow of a clenched fist at the apex, the latter the slap of an open palm. Left ventricular hypertrophy is usually present in cases of patent ductus, ventricular defects, aortic stenosis or coarctation and right ventricular hypertrophy in pulmonary stenosis Fallot's tetralogy and other lesions where the burden falls upon the right side. Further and more conclusive evidence of the predominance of one or other ventricle is afforded by electrocardiography, an indispensable investigation. As well as the character of the apex beat, thrills are palpable in ductus arteriosus, ventricular defects, pulmonary stenosis and about half the cases of Fallot's tetralogy. Auscultation reveals a variety of murmurs which can if necessary be analyzed in more detail by phonocardiography. Useful information is also afforded by close attention to the character of the second heart sound in the pulmonary area. This sound normally has two constituents, first the closure of the aortic valve then that of the pulmonary. If both are audible both great vessels exist and function whereas in Fallot's tetralogy and severe pulmonary stenosis, the sound is single, for only the aortic closure is heard.

Finally must come an estimate of disability of the urgency or otherwise of surgical relief and of the probability of success. Breathlessness is the chief manifestation of congenital cardiac disorder and varies in severity from merely preventing a child keeping pace with his playmates to rendering him immobile and incapable of any effort without distress. The curious but characteristic phenomenon of 'squatting' when a child will suddenly stop and crouch on the

floor to regain breath, is seen in the majority of cases of Fallot's tetralogy, but only in about a quarter of other cyanotic conditions. It is never too early to close a patent ductus, but anastomotic operations should be postponed, if the condition of the child permits, until it is at least 4 or 5. After the age of 20 the chances of success again dwindle in cyanotic disease, and age adds to the risk of surgery for coarctation.

In no branch of surgery is close liaison between physician and surgeon more essential than in this, and the surgeon himself must in the best sense of the term be an operating physician.

CYANOTIC DEFECTS AND THEIR SURGICAL TREATMENT

i. Fallot's tetralogy

The great majority of 'blue babies' (more than 60 per cent.) suffer from this complicated defect which, as its name implies, has four elements:

- (1) a high interventricular septal defect;
- (2) pulmonary stenosis, usually of the infundibular type;
- (3) an aorta which overrides both ventricles;
- (4) as a result of all these things, right ventricular hypertrophy.

The central cyanosis which inevitably accompanies these anomalies is present from birth or very soon after, unlike that seen in a number of other conditions such as simple pulmonary stenosis with right-to-left shunt through a patent foramen ovale. There is always clubbing of the fingers and toes, and the nail beds are seen to be blue even when the hands are warm.

Breathlessness becomes obvious when the child is old enough to move about, and four-fifths of them characteristically 'squat' on the floor to regain their breath after walking short distances. This feature is much less common, though it still occurs, in patients with other cyanotic conditions which cannot be relieved surgically. The cyanosis in younger children is often quite slight while they are resting but is much more conspicuous after a bout of crying.

The infants tend to be slow to gain in weight and some are poorly developed physically; but mental retardation is rare, and many are both intelligent and spoilt, their disablement having made them the centre of much parental attention and devotion. In spite of the severe handicap of their disease they do not seem to succumb more readily than normal children to intercurrent infections, but like all others with congenital cardiac anomalies, fall ready victims to bacterial

endocarditis—a risk which increases with age and is not diminished by successful surgery. The cyanosis tends to progress with time and bouts of unconsciousness due to cerebral anoxia become more frequent, especially after exertion. One of the first signs of the disease worsening is a tendency for the child to regress in play and to associate with those younger than himself, and this, together with convulsive attacks of anoxaemia, is a danger signal that surgical relief should not be too long delayed. About half the children, if untreated, are likely to die before the age of 7, and less than 10 per cent. survive to 21 but the hazards of surgery are great below the ages of 3 or 4 because the small size of the structures increases technical difficulty, because the lumen of any anastomosis made is so tiny that the risk of thrombosis at it is much greater and because it is very doubtful how far such new channels increase later with the patient's growth. For all these reasons, therefore, surgery should be postponed if possible until the child is at least 4. The optimum age for operation lies between 5 and 7, the risks again increasing and the likelihood of a good result diminishing after 20 but clearly when the surgeon is faced with a child whose condition is deteriorating, whatever the age may be, he must accept the risks and do what he can to bring relief.

Examination of the heart shows it not to be enlarged, and the apex beat is not forcible. In the pulmonary area only a single second sound is heard caused wholly by the closure of the aortic valve the pulmonary element being suppressed because of the lowered pressure in the stenosed artery. A systolic murmur, accompanied by a thrill in about half the cases, is present in the same area. Tauszig says that in Fallot's tetralogy the intensity of the murmur is inversely proportional to the severity of the stenosis. No other murmurs are audible. The jugular venous pressure is not raised. The haemoglobin is increased but there is a disproportionate degree of polycythaemia and the red cell count may be in the neighbourhood of 8 000 000. Electrocardiography shows right ventricular preponderance.

On screening, the normal prominence of the pulmonary artery shadow on the left border of the heart is wanting, so that the border itself looks concave and gives rise to the 'sabat-shaped heart' (X ray 122) but this is obvious only in about half the cases. In the left oblique view there is also a clear 'pulmonary window' (i.e. the area below the aortic arch). These signs are really of less importance than the recognition of diminished vascularity in the lung fields themselves, of small hilar shadows, and of absent pulsation. In 25 per cent. the aorta is seen to be right sided. Elaborate catheterization studies and angiography which are not without intrinsic risks of

their own, are unlikely to elicit anything more of importance, and should be reserved solely for those cases in which the exact diagnosis is in doubt. Even the distinction between valvular and infundibular pulmonary stenosis, which must be made if a direct operation is contemplated, can be decided with more accuracy by direct observation, palpation, and pressure measurements at the time of operation. Nor should an arbitrary choice of the surgical measures to be applied in a given case be made before the heart has actually been inspected, for unforeseen anatomical circumstances may in any event dictate which is most expedient. Good results can be expected in 70 per cent. of patients irrespective of the methods employed, with a mortality of between 5-10 per cent. This is, however, *three times greater if the child is less than three years old*, and the proportion of satisfactory results correspondingly less.

At present a choice must be made between four operations for the relief of Fallot's tetralogy. those of Blalock and of Potts, both of which are designed to increase blood flow to the ischaemic lungs by creating an arterio-venous anastomosis, and Brock's operation of pulmonary valvotomy, or direct attack upon the stenosis under hypothermia.

The effect of the anastomotic operation is to create an artificial ductus arteriosus, and this presumably exposes the heart to the same dangers as does a patent ductus, namely an increased risk of bacterial endocarditis and eventual heart failure. Furthermore, nothing will have been done to relieve the strain on the right ventricle which labours as before against the partially obstructed pulmonary artery; and there is some evidence that the degree of stenosis tends to increase with time as fibrin and platelets are deposited upon it. In the case of Blalock's operation some limit is imposed on the size of the fistula created by the small size of the systemic artery employed; but in that of Potts an opening of almost any size can be made, and if this is misjudged pulmonary oedema and right heart failure are likely to follow swiftly. In spite of these objections, however, anastomotic operations have yielded excellent results over the short term, and *in cases of tricuspid and of pulmonary atresia there is no alternative to them*.

It is a fundamental principle of surgery to relieve obstruction where possible; and in this lies the attraction of direct attack upon the stenosis as advocated by Brock. This stenosis may be at the valve itself, or more proximally in the outflow from the right ventricle (which is called the infundibulum), or in both (see Fig 41). In Fallot's tetralogy it is most frequently infundibular (38 per cent.), is at both

levels in 22 per cent., there is complete atresia of the pulmonary artery in 20 per cent. and only in the remaining 20 per cent. is the stenosis valvular. There is no doubt that in this last group pulmonary valvotomy yields excellent results, but in patients with combined infundibular and valvular or infundibular stenosis alone, direct operation on the pulmonary outflow tract under hypothermia has great advantages and seems likely to replace the other methods. It must be remembered that the stenosis is not the only abnormality present, and the septal defect and overriding aorta remain to burden the right ventricle and one day to bring about its failure. The risk also of bacterial endocarditis remains.

Successful surgery of whatever type, results in great diminution in cyanosis, in polycythaemia, and in breathlessness. The patient is able to play and walk, to go to school, and eventually to earn his living as he never could have done before, but he must continue to live within the strict limitations of his still highly abnormal heart, and the ultimate outlook is poor.

Two conditions which greatly resemble Fallot's tetralogy, and must be distinguished from it, are pulmonary atresia and tricuspid atresia, for in them the chances of successful treatment is at least halved, operative mortality is greater and therefore a much greater degree of disability must be present before surgery is justified.

ii Pulmonary atresia

This is suggested by a continuous murmur of the 'machinery' type in a patient who otherwise appears to have Fallot's tetralogy. This is because there is complete obliteration of the pulmonary artery instead merely of stenosis, and the only way in which blood can reach the lungs is through a patent ductus arteriosus or through enormously dilated bronchial arteries in which the blood flow produces a similar bruit. The pulmonary concavity is even more conspicuous radiologically than it is in the usual Fallot's tetralogy and sometimes the anastomotic web of dilated bronchial vessels round the hilum can be seen. The only possible surgical treatment is by subclavian pulmonary anastomosis for as a rule a lumen does exist in the pulmonary artery beyond the area of atresia but angiocardio-graphy is essential to confirm this before a dangerous operation is embarked upon.

iii Tricuspid atresia

Tricuspid atresia is the only cyanotic lesion in which the pulmonary blood flow is diminished and the left ventricle enlarged.

Sometimes blood reaches the right ventricle from the left through a septal defect, but sometimes both aorta and pulmonary artery rise from the left side alone. In cases with a septal defect the diminished pulmonary blood flow can be augmented by an anastomotic operation; whereas if the pulmonary artery arises from the left ventricle lung vascularity is increased, and surgery is contra-indicated. The distinction is made on the radioscopic appearance of the lung fields, and by angiocardiology, but cardiac catheterization is not of much help.

In *transposition of the great vessels* when the aorta rises from the right ventricle and the pulmonary artery from the left, cyanosis is present combined with a greatly increased blood flow in the lungs. Many infants so afflicted die early, but the lesion in those surviving accounts for about 8 per cent. of cyanotic cases. Some attempts to obtain a freer admixture of blood by creating an artificial atrial septal defect or by anastomosing the azygos vein with a pulmonary vein have been tried but have not as yet proved very successful.

Preparation for surgery

Preoperatively the child becomes familiar with the ward and staff who will look after it subsequently. It is introduced to the oxygen tent or cot in which it will be nursed for the first few postoperative days; and great attention is paid to adequate fluid intake, for dehydration in children who are polycythaemic is especially dangerous and likely to lead either to cerebral thrombosis or clotting at the operation site. Some respiratory infection is common and must be cleared up if possible; but if it does not respond to antibiotics fairly quickly it is better to get on with the operation, penicillin therapy in any case being begun a week preoperatively. Patients are not digitalized. Two or three pints of blood are held in readiness to replace any sudden loss that may occur, but transfusion should be avoided if possible because of the added risk of thrombosis, only 4 per cent. dextrose and $\frac{1}{5}$ normal saline being used, to which 0.4 per cent. procaine hydrochloride may be added, up to 500 mg. an hour, to keep cardiac irritability in check.

In all operations upon the heart adequate oxygenation must be a constant preoccupation. On the whole the heart is an organ which stands manipulation well, and one of the chief reasons why the heart stops is that insufficient oxygen is reaching its muscle through the coronary arteries. Anything that interferes with this supply is likely to cause cardiac arrest, such as a sudden fall in blood pressure. This may even happen during anaesthetic induction due to vasodilation

which reduces the cardiac output, so it is valuable to have continuous electrocardiographic readings which give warnings, by depression of the S-T segment or by bundle branch block, of impending anoxia. The other important thing to remember is that the lesion for which the operation is being performed is the chief impediment to the heart's action, so cardiac embarrassment is an indication to press speedily on with its relief. This is likely to do far more good than cardiac massage or the injection of drugs, though these too have their place.

Blalock's operation

Although Blalock himself originally described and preferred a right-sided approach most surgeons now employ a left thoracotomy because it gives better access to the rest of the heart and so enables a more complete diagnosis to be made, especially regarding the nature of the pulmonary stenosis. It also gives access to the aorta except in the 25 per cent. of patients whose aortas are on the right, so that there is much greater flexibility and a free choice of the best operation to employ in the circumstances revealed by direct examination.

The chest is entered through the whole length of the bed of the fourth rib. The pericardial sac is opened, the anatomy of the heart and great vessels is carefully examined with especial reference to the nature of the pulmonary stenosis present. If necessary direct pressure readings are made by passing a cannula into the right ventricle and the pulmonary artery. The presence of a normal aorta and its branches is confirmed, for if Blalock's operation is to be performed the subclavian must be of sufficient length to reach the pulmonary artery after division and the pulmonary artery itself must be a vessel of sufficient calibre for anastomosis to be practicable. It is at this stage therefore that the final choice of operation is made.

The lung is retracted downwards to expose the superior mediastinum. This is done gently and from time to time the lung is released and reinflated to secure maximum oxygenation. The mediastinal pleura is divided, anastomotic vessels underlying it are carefully tied and cut, and the pulmonary artery is exposed. The fine areolar sheath covering the vessel is dissected off so that the artery wall is perfectly clean from its origin to a little beyond its first division. The vessel is occluded for a few minutes to see whether the opposite pulmonary circulation maintains adequate oxygenation. If all is well the subclavian artery is dissected free from the tissues of the mediastinum. When a right-sided aorta is present the left subclavian is

a branch of the innominate, and special care must then be taken not to injure the recurrent laryngeal nerve where it hooks round the artery to reascend into the neck. The superior intercostal vein crosses the artery superficially and is divided between ligatures. The subclavian is freed up to its vertebral and internal mammary branches, and the operator must be satisfied that there is a sufficient length of the vessel to turn down to the pulmonary artery without tension or sharp angulation. A little extra length may sometimes be gained by flexing the child's head on to its left shoulder, or by cutting the inferior pulmonary ligament. If possible it is better to divide the artery below its branches, so that anastomotic channels to the arm are not disturbed, but very often it is necessary to ligate the branches and divide the subclavian at their point of origin in order to obtain a slightly wider funnel-shaped end for the line of anastomosis. A small bull-dog clamp is applied at the aortic end of the artery before it is divided distally. The cut end is scrupulously cleaned of all areolar tissue.

A ligature is now tied round each end of the bull-dog clamp. The ends (which are left long) are passed underneath the pulmonary artery and used by the assistant to draw the subclavian downwards and into close apposition with it. Blalock's clamps are placed across the pulmonary artery as far proximally and distally as possible to leave the maximum area of vessel clear for suturing. These clamps are steadied, and kept straight, by one assistant only (Fig. 39).

An incision to match the available lumen of the subclavian is made in the pulmonary artery wall, a stay suture being placed at each extremity of the incision, and left untied. A posterior everting 4 or 5/0 arterial suture on an atraumatic needle is then inserted beginning at the first stay suture, and passing from the outer to the inner side of the subclavian, thence to the inside of the pulmonary artery, back to the outer side of the subclavian, and so on. When the row is completed the suture line is gently pulled tight approximating the two vessels, and the ends are tied to the stay sutures which are tied in turn. All knots are tied externally, and each bite is 1 to 2 mm. long. The anterior edges are similarly sutured, or a continuous everting mattress stitch may be employed. When the anastomosis is complete the distal clamp on the pulmonary artery is released and any oozing at the suture line controlled by gentle pressure, or, if necessary, by inserting extra interrupted stitches where required. The proximal pulmonary clamp is taken off, and finally the bull-dog clamp on the subclavian, the anastomosis being inspected on each new accretion of pressure. As soon as free flow through the junction is established a thrill becomes palpable.

If the pulmonary artery is unusually small though not actually atresic, or if it is found undue tension is needed to anastomose the

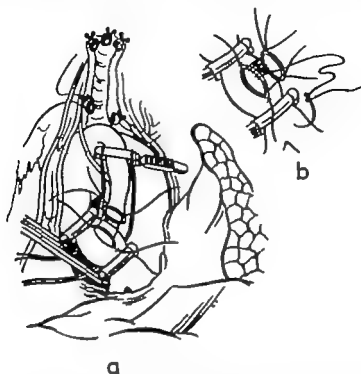


FIG 39 *Blalock's operation*

(a) The left subclavian artery has been divided from its branches and turned down from the aortic arch to meet the pulmonary artery which is occluded by two Blalock clamps. The subclavian is held in position by two sutures tied to the bulldog clip near its origin and passing behind the pulmonary artery. The wall of the latter has been incised.

(b) The anastomosis between the cut end of the subclavian and the side of the pulmonary artery

subclavian to its side, the pulmonary artery may be tied and divided as it emerges from the pericardium and an end-to-end junction made between its distal part and the subclavian artery

Potts' operation

Provided the aorta is on the left side an aorto-pulmonary anastomosis can be performed as an alternative to Blalock's operation. The pulmonary artery is dissected free and the ability of the child to survive its temporary occlusion tested as in Blalock's operation. A pair of fairly thick silk ligatures are placed to encircle the main trunk close to its origin and close to its first branch, so that by pulling upon them flow through the segment between them is abolished

(Fig 40a) They are left untied. A part of the descending aorta to which the pulmonary artery can easily be approximated is chosen, and after the pleura and fascia overlying it have been stripped off, it is mobilized by division of two or three pairs of the related intercostal arteries. A Potts clamp of suitable size is used to isolate a segment of the aortic wall without interrupting the flow through the aorta itself. If necessary the part of the wall between the jaws of the clamp is pulled out with forceps to give more room for the anasto-

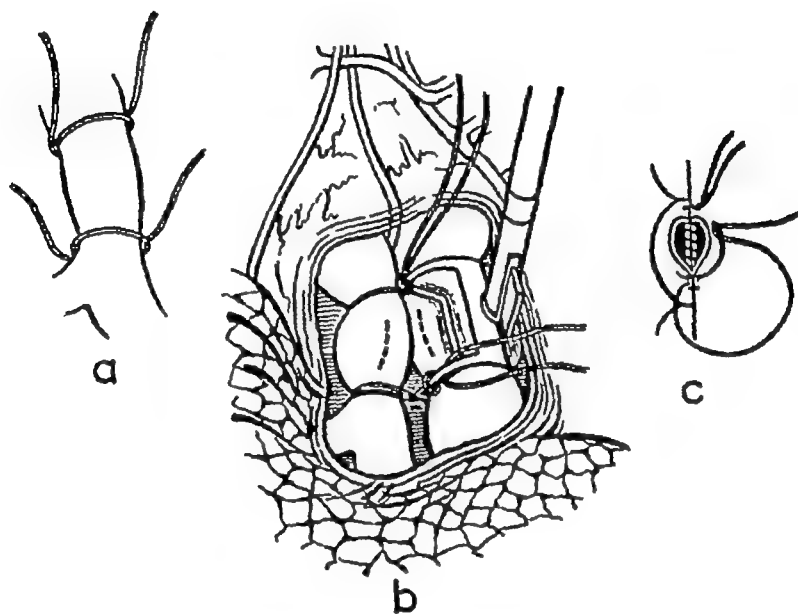


FIG. 40 *Potts' operation*

(a) The left pulmonary artery is encircled by ligatures which will occlude it temporarily when they are tied to the Potts clamp

(b) The Potts clamp is applied to the aorta isolating a segment of its wall for anastomosis but not interrupting the main blood flow, and the pulmonary artery is tied to it by the ligatures seen in (a). Matching incisions are made as indicated by the dotted lines

(c) The anastomosis.

mosis. When the clamp is closed a thrill is felt distal to it confirming that the main blood flow continues, and this is important, for if the aorta is completely occluded the spinal cord is likely to be deprived of its blood supply

The ligatures round the pulmonary artery are now tightened and tied firmly to each end of the Potts clamp, thus both occluding the pulmonary artery, and bringing it into close contact with the aorta (Fig. 40b). Incisions not more than 4 mm. in length, cut squarely with a very sharp knife, are now made through the wall of the aortic

lip held by the clamp and the corresponding part of the pulmonary artery. If this length is exceeded the resulting fistula will be too great and almost inevitably lead to heart failure. 4 or 5/0 vascular sutures on a No. 9 curved atraumatic needle are then used for the anastomosis (Fig. 40c). The stitch begins at the upper angle and is carried through the wall of the pulmonary artery from outside in, across through the aortic wall from inside out, and is tied so that the knot is external. The posterior edges of the orifices are sutured together by a continuous over-and-over stitch with bites about 1 mm. apart, and when the lower angle is reached the suture is locked or tied, and the anterior layer of the anastomosis similarly completed. First the distal and then the proximal ligatures occluding the pulmonary artery are cut and slipped off, and the anastomosis tested for leaks under the increasing pressure. Its posterior aspect is inaccessible, so the greatest care must be taken with its suturing in the first place. Finally the Potts clamp is removed from the aorta. A marked thrill is at once felt over the pulmonary artery as the systemic blood courses into it.

iv Pulmonary stenosis

Pulmonary stenosis may exist as an isolated deformity and may fall either into the cyanotic or the acyanotic group. The presence or absence of cyanosis depends in the main on whether or not there is also a defect (such as a foramen ovale) in the interatrial septum through which a right to-left shunt of blood can pass when the pressure begins to rise as the right ventricle labours against the obstruction to its outflow.

The consequence of this mechanism is that these children are not cyanosed in the early months of life as are those with Fallot's tetralogy, but cyanosis appears only after some years, and often not until late adolescence. For the same reasons there is not the same degree of breathlessness in infancy; the children do not 'squat' or lose consciousness and gain weight quite satisfactorily in contrast to those with Fallot's tetralogy who tend to be poorly developed. In a great many cases stenosis is mild, the prognosis excellent, and surgery is not required.

On the other hand when right ventricular strain begins it is likely to be more swiftly progressive and more serious because in a Fallot's the overriding aorta provides a kind of safety valve. The result is that the Fallot heart remains small whereas that of the pure pulmonary stenotic rapidly enlarges. In patients with Fallot's tetralogy the chief problem is one of anoxia; in those with pulmonary stenosis

It is right heart failure. Direct relief of the stenosis is therefore indicated in preference to an anastomotic operation.

Pulmonary valvotomy was first performed by Brock and it is to him that we are indebted for the development of the techniques now employed. Fortunately the type of stenosis (again in contrast to that found in Fallot's tetralogy) is almost always val-

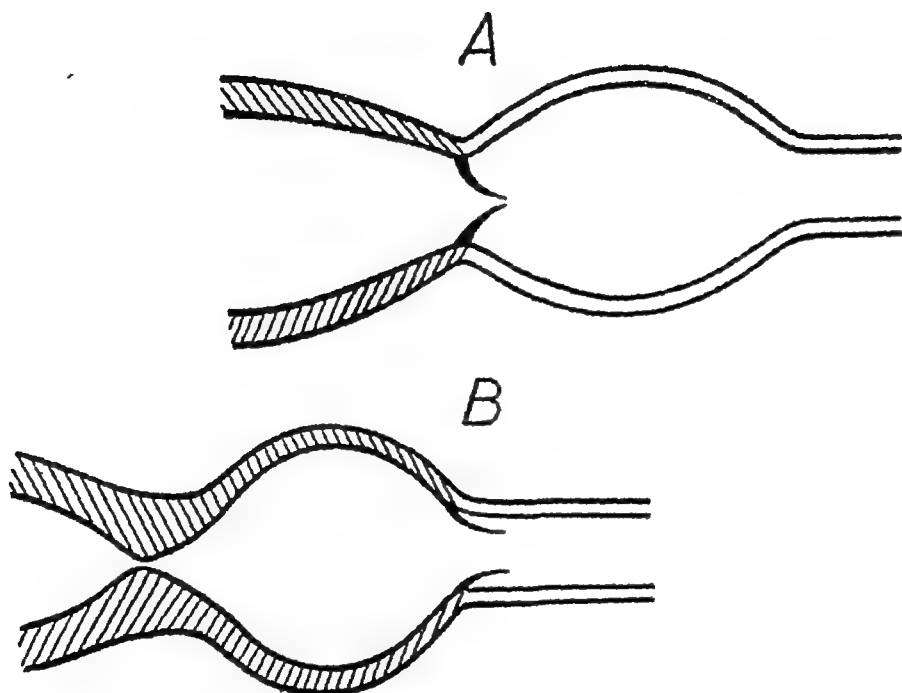


FIG. 41

Diagram of the difference between valvular and infundibular pulmonary stenosis

(a) Valvular. The muscular outflow tract is normal and there is post-stenotic dilatation of the artery. This is the type best suited to valvotomy.

(b) Infundibular. Here the stenosis is not at the valve but in the myocardial wall of the outflow tract itself, a lengthy segment being affected. This is clearly much less suitable for instrumentation.

vular and so lends itself better to valvotomy (Fig. 41). Not all pulmonary stenotics require surgery for there is much variation in the degree of obstruction; but the lesion accounts for some 6 per cent. of patients in the cyanotic, and 20 per cent. of those in the acyanotic group, and is therefore as common as patent ductus arteriosus.

When no evidence of right ventricular strain is apparent, either clinically or on the electrocardiogram, surgery is not indicated. In

the remainder jugular venous pressure is likely to be raised although pulse and apex beat remain small, and a harsh systolic murmur and thrill are present in the second or third left intercostal spaces. In this position the second heart sound is both single and diminished. Screening shows the right ventricle to be enlarged, and although the lung fields themselves are ischaemic the pulmonary artery is dilated beyond the stenosis, and the border of the heart is not concave as it is in Fallot's tetralogy. Surgery is more dangerous and the result less successful when cardiac enlargement and cyanosis have begun, the decision to operate should therefore be made early rather than late. Cardiac catheterization is of value in detecting the rise in right ventricular pressure before it is clinically manifest. A pressure of 80 to 100 mm. of mercury is an indication for surgery, as is electrocardiographic evidence of increasing right-sided strain. Valvotomy should reduce the ventricular pressure by more than half. The chief risks of the operation are sudden cardiac arrest or failure. These are more likely to occur in patients who have reached their teens or twenties, and whose hearts are big, than in younger ones in whom disability and cyanosis are still slight.

Pulmonary valvotomy

The patient lies supine with left arm extended laterally on a splint. The left chest is entered through a curved infra mammary incision in the third interspace, from mid-axilla to sternal edge. After the pericardium has been widely opened and the diagnosis confirmed by inspection and palpation of the pulmonary valve (beyond which there will probably be seen post stenotic dilatation of the artery), a point is selected on the anterior wall of the right ventricle some 5 cm. below the valve, and a short incision made almost through the myocardium, which is finally pierced by a curved probe. This is felt between left index and thumb as it is passed up into the stenosed valve. Succeeding instruments are similarly guided but care is necessary at all times not to occlude blood flow in the artery either with instruments or by pressure. A cardiac catheter is inserted, and the pressure gradient across the stenosis measured. After valvotomy these readings are repeated to ensure that relief has been given and if results are unsatisfactory dilatation must be repeated. The valvulotome probe-pointed but with sharp shoulders is next passed, the probe engaged in the stenosed orifice, and the cusps cut across by a little pressure. An expanding dilator finally ensures complete freeing of the stenosis, and leaves behind two cusps which do not permit

regurgitation The wound in the ventricle is closed by two or three fine silk sutures through the myocardium.

Open valvotomy and infundibular resection

This operation has become possible only with hypothermia; and because it permits direct inspection of valve and infundibulum, and both may be efficiently dealt with when both are stenosed (see p 292), it offers great advantages over indirect valvotomy (which is unsatisfactory in cases of infundibular stenosis), and over the anastomotic operations for Fallot's tetralogy, and it is now tending to supplant them all as the most satisfactory treatment for the deformity.

The patient is cooled to 30° C (see p 285), and lies on the table with both arms extended on splints. Through a curved bilateral infra-mammary incision reaching from axilla to axilla, the right pleural cavity is opened in the fourth intercostal space, the left in the third, the sternum between them cut across, and the thoracic contents widely exposed. The whole front of the pericardium is turned back as a rectangular flap and stitched to the edge of the right thoracotomy wound thus covering and restraining the right lung. Tapes are passed round both superior and inferior venae cavae, and threaded through rubber tubing to be used later as tourniquets. The pulmonary outflow tract is examined and the diagnosis confirmed. A stay suture is placed in the myocardium just below the infundibulum, and another in the artery wall just beyond the valve. The venae cavae are occluded, ten or twelve beats allowed to empty the heart, and a clamp applied in the transverse sinus to close the aorta and pulmonary artery, 0.03 mg. of neostigmine per stone of body weight being injected into the aorta proximal to the clamp. The pulmonary outflow tract between the stay sutures is incised, the valvular stenosis (if one is present) cut with scissors to give two valvular cusps, and the myocardium obstructing the infundibular channel bitten out with a small bone rongeur. The inferior caval ligature is gently released to flood the field with blood (eliminating the risk of air embolism), and the myocardium and artery wall closed with a running suture tied to the stays at either end. Both venae cavae are then fully released, and after the heart fills, the clamp is removed from the transverse sinus. The pericardial flap is sewn back into place, the lungs re-expanded, the sternum wired together, and the two thoracotomy incisions closed, a water-sealed drain being left in each pleural cavity.

The cooling process ceases at the beginning of the operation, and the temperature slowly begins to rise again towards its end. Active

re-warming (by the circulation of warm water through the coils) is then begun and the temperature raised to about 35° C before the patient is returned to bed.

Postoperative care

After all these operations—those of Blalock and Potts as well as pulmonary valvotomy—a water-sealed drain is left in the pleural cavity for 24 or 48 hours, both to ensure full re-expansion of the lung and to enable serious bleeding to be detected promptly. Patients are returned to bed in an oxygen cot or tent where they are nursed flat until fully conscious and kept during the first few days. This is the most critical period, during which heart failure, pulmonary oedema, cerebral thrombosis or clotting at the operation site are most likely. The pulse rate and blood pressure are at first recorded quarter hourly so that any sudden fall in pressure can be counteracted by small doses of suitable vasopressor drugs, and if necessary by transfusion. Handling of the child is apt to cause such falls in pressure and should therefore be avoided.

Constant attention is devoted to maintaining good aeration. The mouth and pharynx are kept clear, if necessary the trachea is sucked out, and air or pleural effusion impeding the expansion of the lung removed. Rest is enforced and sedatives given as may be needed to secure it. Any sign of cardiac enlargement is an indication for the continuance of rest and this must also be watched for much later when convalescence begins for it is then that new-found freedom is most likely to be abused and too much activity may throw an excessive burden upon the heart.

Antibiotics are continued for the first postoperative week. It is often noticeable that wound healing takes longer than usual and superficial infections are common due presumably to deficient nutrition.

In the days following operation there is generally a prompt improvement in the patient's colour and the haemoglobin and red cell count fall sharply. Radiologically increased vascularity of the lung fields is seen and this may be excessive especially after Potts operation and accompanied by some degree of cardiac enlargement calling for prolonged bed rest.

ACYANOTIC DEFECTS AND THEIR SURGICAL TREATMENT

The incidence of pulmonary stenosis with an intact septum in this group has already been described. The other main conditions are

patent ductus arteriosus; coarctation, aortic stenosis; and atrial and ventricular septal defects. The surgical cure of most of these is now a standard proceeding; but that of ventricular defects is less well defined and remains largely in the sphere of experimental surgery, although the number of successful results is slowly mounting. In septal defects and patent ductus arteriosus the essential lesion is a left-to-right shunt of blood; in the others it is obstructive.

Coarctation and patent ductus are of course both defects of aortic arch development and lie outside the heart itself; but whereas certain other anomalies of the great vessel exist and manifest themselves in other ways, the burden of these two falls primarily upon the heart.

i. Patent ductus arteriosus

During intra-uterine life venous blood from the right heart short-circuits the unexpanded lungs to reach the aorta and the placenta through the ductus arteriosus, a short wide channel which connects the left pulmonary artery directly to the medial side of the aorta. When the lungs expand at birth the ductus normally closes very rapidly, probably partly by contraction of smooth muscle in its wall and partly due to the sudden change in blood flow and pressure; and it becomes the sclerosed *ligamentum arteriosum*. For reasons that are obscure it sometimes remains patent (three times more often in girls than in boys), and it then constitutes an arterio-venous fistula through which serious leakage of blood occurs from the aorta into the pulmonary artery. This leakage varies, of course, with the size of the ductus, but it sometimes amounts to more than half the output of the left ventricle, with the result that the latter enlarges and eventually fails. In addition, the impact of the systemic pressure falling upon the pulmonary artery and the lungs leads to dilatation of the right ventricle and to pulmonary hypertension. Cyanosis is never present unless a reversal of flow has taken place under these circumstances so that blood flows from the pulmonary artery into the aorta instead of in the reverse direction. This reversal, and the cyanosis accompanying it, are exceedingly rare. No 'machinery' murmur is present; and the diagnosis calls for the most rigorous investigation before it can be accepted. A patent ductus sometimes accompanies other congenital cardiac lesions, when its presence may be life-saving if it enables blood to reach the lungs in the same way as does the artificial fistula created by anastomotic operations. Under such circumstances it must not be tied.

The ultimate result of a patent ductus is likely to be death from left heart failure. Very few patients survive much beyond the age of

30 and those who do have a very small ductus, for the lease of life depends on the size of the leak. In addition there is serious risk of bacterial endocarditis supervening. This occurs in about 30 per cent. of patients, and although infection can often be controlled by antibiotics, it remains a grave disease with 20 per cent. mortality. The chances both of cardiac enlargement and of infection increase progressively with age, so the sooner a ductus can safely be closed the better. It is often said that the risk of bacterial endocarditis under the age of 6 or 7 is negligible and that it is never seen in smaller children. This is not so. A little girl aged 4 years who was known to have a patent ductus was referred to me very seriously ill. She had had a high fever for several weeks and was lethargic and sallow. Her spleen was palpable, there were petechiae scattered over her abdominal wall and as well as the characteristic signs of patent ductus she had a rapid thrready pulse and severe anaemia. A blood culture grew *Streptococcus viridans* in twenty four hours. After prolonged and massive dosage with penicillin she recovered and I ligated her ductus. She made a good recovery.

This case demonstrates that it is not safe to rely on the hope that small children will not contract endocarditis. Once the diagnosis is established there is no excuse for procrastination. The absence of symptoms indicates a favourable opportunity for surgery and is not a contra indication to it. There are other potent reasons. A boy of 11 was presented at his mother's request because she had read so much about the heart operations they are doing now. When he first went to school five or six years previously an abnormal cardiac murmur had been heard by the school doctor and the boy forbidden to swim or to play games. This ban was greatly reinforced by his mother who was told simply that he had heart disease and must take things quietly. From that time on he was never allowed to play with other children at the least indisposition was kept at home for long periods and spent most of his day on a *chaise-longue*. For no good reason he had been robbed of most of his primary education and much worse, his childhood. His ductus was ligated and at the end of a fortnight his mother and his school, were instructed that his heart was now normal and no checks were to be put on his activities. This case history although extreme could be matched by many others. Even were there no risk of ventricular enlargement or of endocarditis, the limitation which is at once put upon these children's lives when they are found to have a loud heart murmur condemning them to years of quite unnecessary cardiac invalidism, would be an indication for early surgery.

A patent ductus in infancy as a rule produces no symptoms except that the child may become breathless more quickly than others if the communication is large, and may appear to suffer from chronic bronchitis because of resulting pulmonary congestion. The physical signs of patent ductus arteriosus are often first detected on routine examination at home or in a school clinic. A systolic thrill is felt, maximal over the second left interspace, and it is accompanied by a machinery-like murmur which is continuous throughout systole and diastole. Such findings are in themselves virtually diagnostic and are present in 95 per cent of cases. Very rarely such a thrill and murmur may accompany a fistula in the aorto-pulmonary septum, and in some instances of very wide patency the murmur is discontinuous or absent, but cardiac catheterization (which is quite unnecessary in ordinary cases) is needed to confirm the latter, and a diagnosis of aorto-pulmonary fistula could not be made with any assurance before thoracotomy.

The pulse is of the water-hammer type because of much increased pulse-pressure, the systolic tension being normal, the diastolic lower than usual. The heart is not often enlarged in young children, but if the shunt is great, or the diagnosis delayed to later life, it may be very large. Radiographically the lung fields look congested, the pulmonary artery shadow is prominent, and hilar pulsation increased. These signs also accompany an atrial septal defect which must be distinguished from a patent ductus for it is a common congenital defect. Although a thrill may be present in the pulmonary area in septal defects, the murmur that accompanies it is systolic only, never continuing through diastole, and the atria are themselves enlarged, as they are not in patent ductus. If doubt exists as to whether the murmur heard is in fact continuous or not, it is studied by phonocardiography. The electrocardiogram either shows no abnormality, or varying degrees of left axis deviation.

The onset of *bacterial endocarditis* is unusual before early puberty, though infants are by no means immune. Insidious at first, the infection is accompanied by irregular fever, increasing weakness, anaemia, pallor and loss of weight. There are often evening rigors and sudden pains in joints. The diagnosis is proven by the growth of *Streptococcus viridans* on blood culture, but not infrequently culture is negative because the organisms are filtered out in the lung parenchyma. For the same reason embolic phenomena tend to be late, and may not be present at all, although blood cells in the urine, petechiae, transient painful nodules, or haemoptyses must all be watched for. The spleen is sometimes palpable. Untreated, the disease

progresses over several months to a fatal termination, but it can be brought under control by massive antibiotic therapy. If this is successful a period of recuperation should precede ligation of the ductus. Ligation is in any event strongly indicated, and closure has been demonstrated to sterilize the blood stream within an hour, though this can be relied upon only in 60 per cent. of cases. Infection substantially increases surgical risk, for the ductus wall may be much more friable. Prevention in such circumstances is infinitely better than cure. *The patent ductus ought to be closed early in childhood at the end of the first, or during the second year when risk is least and disastrous complications have not occurred.*

Surgical closure

A left postero-lateral incision is made through the fourth intercostal space without removal of the rib. The lung is retracted gently downwards, and the heart and region of the ductus inspected and palpated. The maximal thrill is usually not over the ductus itself but over the base of the adjacent pulmonary artery. The pleura covering the superior mediastinum is opened in the triangle formed by the phrenic and vagus nerves. In this triangle, just above the lung root, lies a constant lymphatic gland. When this gland and the adjacent fatty and areolar tissue are dissected away the ductus is exposed with the recurrent laryngeal branch of the vagus curving round its inferior border to re-ascend posterior to it, behind the arch of the aorta. At all times great care is taken to protect this nerve from injury and it should not be interfered with, merely recognized and avoided.

At first the ductus seems shorter and wider than it really is, because of a diverticulum of the pericardial sac which overlaps its lateral and superior surfaces. This is peeled back by sharp dissection, and a reflection on to the aorta of the fibrous pericardium, which obscures the upper edge of the ductus is cut with scissors (Fig. 42). As its anatomy is defined, the ductus is almost always found to be longer and narrower than it appeared at first.

A right-angled O'Shaughnessy forceps is introduced under the inferior margin of the ductus and by very gentle blunt dissection is passed up behind until the tips emerge in the superior gap. Pressure between a finger and the forceps abolishes the thrill and occlusion is maintained for a few minutes while the child's colour and condition are observed in order to be sure that the channel is not compensating for some unsuspected cardiac lesion.

A skein of floss silk is then grasped by the forceps, drawn round

the ductus, and firmly tied as closely as possible to its aortic end after making sure that the recurrent laryngeal nerve is safe (Fig. 42). Both the thrill, and the cardiac turbulence previously seen, should be completely abolished. Persistence of a thrill means either that the knot has been tied insufficiently tightly, or that some other lesion is present. A second skein is similarly passed and tied on the side of the pulmonary artery. If ligation is efficiently carried out there is

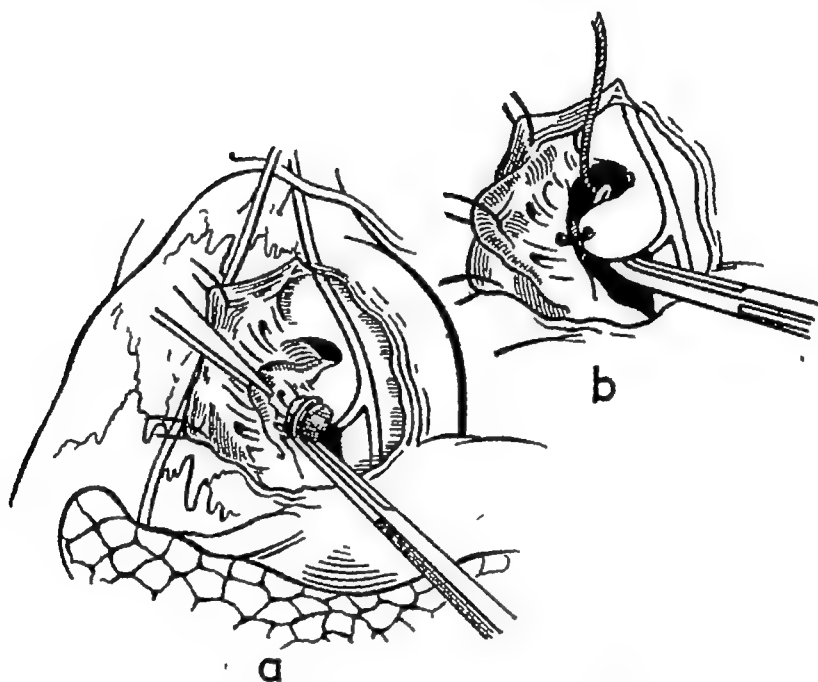


FIG. 42 Closure of patent ductus arteriosus

(a) The overlying pericardial flap is dissected from the ductus, round which passes the recurrent laryngeal nerve

(b) Two thick skeins of floss silk are carried round the ductus by an O'Shaughnessy forceps and tied at either end.

no danger of recanalization (which sometimes occurred when solitary or finer ligatures were used). The mediastinal pleura is closed by a continuous stitch and the lung re-expanded. Care is taken that all parts of the lung are well aerated before the chest is closed. No drain is necessary.

Should the ductus be exceptionally short and wide, making ligation dangerous, or should it be damaged during dissection, complete division with suture of the cut ends is necessary. Suitable clamps should always be available. Bleeding from the ductus is most likely to occur from its deep surface and can be controlled by direct digital

pressure upon it while one Potts clamp is applied to the aorta, and another to the pulmonary artery. The ductus can then be divided in the middle and the cut ends closed with a continuous arterial suture of 4/0 silk. Alternatively two pairs of special Gross ductus clamps (which have very narrow jaws with fine teeth) are applied to each end of the ductus after it has been completely dissected. The two middle clamps are then removed, the intervening flange of tissue cut, and its ends oversewn as before. The remaining clamps are taken off any leakage dealt with by inserting one or two additional interrupted sutures, and Oxycel or coagulant foam packed over the suture lines until all oozing ceases.

On the first postoperative day the child is encouraged to get out of bed and an X ray at this time is taken to confirm full expansion of the lung. Only rarely does a small effusion require aspiration but atelectasis calls for prompt bronchoscopy. The pulse pressure becomes normal as soon as the ductus is tied. As a rule no murmur remains, but sometimes a systolic bruit persists in the second inter space. It is of no importance, and all concerned must be impressed with the fact that the child is now wholly normal and can lead an active and unrestricted life henceforth.

II Atrial septal defects

Atrial defects are among the commonest of congenital anomalies but generally do not cause trouble, in spite of the heart being large until in middle age the patient begins to suffer from increasing breathlessness due to the shunt of blood from the left to the right atrium, and ultimately succumbs to congestive failure. It is the only congenital condition in which atrial fibrillation is common but this too is a late phenomenon. The patient is usually recognized by his having much less disability than would be expected for the size of his heart. The pulse is small, the apex beat sometimes tapping, but sometimes more forceful due to right ventricular hypertrophy and a soft systolic murmur is audible in the pulmonary area and may be accompanied by a thrill. The pulmonary second sound is widely split. The electrocardiogram shows right bundle branch block. Radiographically the right side of the heart and the pulmonary artery are conspicuously large the left side and the aorta small. The vascularity of the lung fields is increased (X ray 127). Cardiac catheterization is of value in establishing the diagnosis, when the catheter is found to pass from right to left atrium and a shunt is demonstrated. Most interatrial defects lie posteriorly (ostium secundum) and can be sutured without interfering with other structures, but some, the

so-called ostium primum group, are anterior, and so low down that they affect the atrioventricular valves and cause mitral or tricuspid incompetence. As the lower edge of the hole is virtually constituted of the valves themselves ostium primum defects present a much more difficult problem than the commoner variety, and every effort should be made to recognize them clinically—by the presence of mitral or tricuspid incompetence—and reject them for surgery.

Because of the poor ultimate outlook, and because an arterio-venous fistula of this kind pre-eminently lends itself to surgical repair, operative closure under hypothermia should be advised in all cases where cardiac catheterization shows the shunt of blood to be significant. There is now no place for the blind procedures (such as that of Bailey) formerly practised.

After anaesthesia the patient is cooled (p. 285) until the oesophageal temperature is 32°C . Cooling is then stopped and the operation begun, for the temperature continues to drop to about 30°C . The position on the table, and the incision, are exactly as already described for the operation of infundibular resection (p. 302), except that both right and left pleural cavities are entered through the fourth intercostal spaces. The pericardium is opened and stitched back to give good exposure of the enlarged right atrium, and tapes are passed round superior and inferior venae cavae. A special clamp is applied to the right atrial wall and a finger introduced through a small incision so that the diagnosis can be confirmed, and the caval orifices and mitral and tricuspid valves palpated. If the defect is of ostium secundum type, the venae cavae are occluded, the aorta and pulmonary artery clamped in the transverse sinus (neostigmine being injected as before), and the right atrium widely opened. The defect is rapidly closed with a continuous stitch, beginning at its lower extremity, where care is taken to see the inferior vena caval orifice is not displaced into the left atrium. The anaesthetist by gentle pressure keeps pulmonary blood from the left atrium welling up in the defect until it is shut, lest air enter the left heart and cause embolus, and for the same reason the right atrium is filled from the inferior vena cava before the clamp on its wall is reapplied. Both cavae are then released, followed by the aorta and pulmonary artery, the atrial wall is sutured, the clamp on it removed, and the pericardium and chest closed as before (p. 302).

The chief hazard of the operation, apart from technical difficulties in stitching the defect, is the risk of air embolus. Should air bubbles be seen in the coronary vessels vigorous cardiac massage is indicated. Surgical results are good, mortality in skilled hands very low.

III Ventricular septal defects

These are often situated high on the septum in a relationship to the valves not unlike that of the septum primum interatrial defect, and so would be difficult to close effectively even if access to them were easy. They occur both as isolated lesions (about 8 per cent. of all cases of congenital heart disease) and in conjunction with other deformities such as Fallot's tetralogy. In the former a harsh systolic murmur with a thrill is present just to the left of the sternum in the third or fourth interspaces. Radiologically the heart tends to be globular in appearance and there is pulmonary congestion. The most successful method so far developed for closing them is that of Lillehei who uses a donor cross-circulation and opens the dry right ventricle so obtained to suture the deficiency under direct vision.

In 25 patients so treated he had 7 deaths, none of them in the theatre, and the 18 survivors did well. In 10 others with Fallot's tetralogy he closed the defect and resected an infundibular pulmonary stenosis with 4 deaths.

IV Coarctation of the aorta

Aortic stenosis and coarctation together account for about a fifth of congenital heart conditions without cyanosis. Coarctation (which has been estimated to occur once in 7 000 births) is four or five times commoner in boys than girls, and is in this respect unlike most other congenital cardiac defects. A rather artificial distinction is made between infantile and adult types of coarctation. In the infantile variety a long segment of the aorta is obliterated, from the subclavian artery down to the ductus, which is widely patent, and through which blood from the pulmonary artery reaches the descending aorta. If the ductus closes, the condition is incompatible with life and as it is in any case often associated with other congenital defects long survival is exceptional—hence the adjective infantile. In the adult type (with which we are here concerned) the constriction is usually, but not always limited to a short segment near the point where the ductus, or ligamentum arteriosum, joins the aorta (Fig. 43). It seems probable that coarctation originates as part of that sclerosing mechanism which occurs just after birth in association with closure of the ductus, the process going too far—or being misdirected—for in 10 per cent. of cases of coarctation the ductus remains patent. The constriction is usually severe, and the aortic lumen is sometimes reduced to the diameter of a pin's head, most of the blood reaching

the lower half of the body through anastomotic channels. In infancy and early childhood there are often no symptoms, making the decision to perform a severe and dangerous operation difficult. Most patients, however, develop symptoms before the age of 25, the average age at death is 32, and 90 per cent. are dead before they reach 50.

About one quarter live out a reasonably long life with little or no disability; about a quarter die, usually in the third decade, from bacterial endocarditis or aortitis, another quarter from rupture of the aorta; and the rest from the effects of hypertension, some from congestive heart failure, some from cerebral haemorrhage. Rupture of the aorta and cerebral haemorrhage are commonest in the second and third decades, death from left ventricular failure tending to be somewhat later. It will be seen therefore that the outlook is bad in 75 per cent. of patients, and as no means exist of detecting in infancy which will be the lucky 25 per cent., the arguments in favour of early surgery are very strong. A limiting factor, however, is the fact that in 25 per cent. of cases the aortic valves are bicuspid and may be associated with a degree of aortic incompetence which constitutes the chief burden upon the heart. In another 10 per cent. berry aneurysms of the Circle of Willis and other cerebral vessels exist which may still rupture after successful surgery; although the risk seems to be diminished.

The longer the coarctation remains, the worse are the arteriosclerotic changes resulting from hypertension, and such changes have even been observed in a child of $3\frac{1}{2}$. It follows that in older patients the arterial wall may be unsuitable for suture, and irreversible changes in the terminal arterioles may have occurred preventing reduction of the blood pressure even after operation. In addition the collateral circulation becomes a more and more formidable surgical obstacle as time goes by.

If a patient survives to 30 surgery should probably not be advised; and 35 represents the extreme upper age limit. Below 5 the infant thorax is inconveniently small, and below 10 doubt remains as to whether the lumen of the anastomosis made will increase adequately, or at all, with age. The ideal time for surgery therefore is between 10 and 15, but clearly this must be disregarded in the face of worsening symptoms, or evidence of increasing left ventricular strain. The natural prognosis is said to be better in girls than boys, so in them a watchful policy is more justifiable.

At any age resection and anastomosis of the aorta is a lengthy, dangerous and difficult operation which ought never to be under-



121

ray 121. An angiocardiogram showing infundibular pulmonary stenosis. Just below the aortic knuckle, there is a post-stenotic dilatation of the pulmonary artery. Immediately inferior to which is the narrowed segment.



122

ray 122. The typical 'sailor-shaped' heart of Fallot's tetralogy.



123

ray 123. An aneurysm of the aorta proximal to coarctation. The patient was a woman aged 30. Calcification is already present in the aneurysm wall. There is notching of the inferior borders of some of the ribs seen in the 5th on either side.



124

ray 124. A large aortic aneurysm has compressed the bronchus to the left upper lobe. Infection has occurred in the obstruction and an abscess with a fluid level is visible to the left of the aneurysm.



125

125 Heavy calcification round the left ventricle in a case of constrictive pericarditis



126

126 An oblique view of the same heart seen in X-ray 125 Note how much calcification lies inferior to that the latter lies in it as in a cup



127

127 Atrial septal defect A cardiac catheter enters the enlarged right atrium by the superior vena cava through the defect into the left atrium before reaching the left superior pulmonary vein Vascularity of lungs is increased



128

128 Pulmonary oedema in a patient with mitral stenosis

taken except under the best conditions and by a specially skilled and experienced thoracic surgeon. In these circumstances a mortality rate of about 7 per cent. is to be expected with good results in the great majority surviving.

Boys with coarctation are usually sturdy and well built and the rarer girls are said by Taussig to show precocious development, suggesting that the condition may be caused by hormonal influences. In early life there are often no symptoms but headaches, flushing, oppressive throbbing in the head and neck made worse by stooping, and breathlessness appear later and may be present very early. The diagnosis depends essentially and simply upon the recognition of raised blood pressure in the upper limbs, and a lowered one, with absent or much diminished femoral and popliteal pulses, in the legs. Unusual pulsation is often visible in the neck and the degree of hypertension found in the arms varies with age and the severity of the lesion from slightly above normal to one in which the diastolic pressure is above 100 mm. and the systolic pressure is over 200 mm. of mercury. Exercise produces an abrupt increase—often of 100 mm. or more—in these pressures. Should a marked difference (i.e. of 30 or 40 mm. of mercury) be found to exist between the two arms, it indicates that the stenosis lies *proximal* to the left subclavian and is therefore probably inoperable. In the lower limbs the pressure (which is normally 10–20 mm. of mercury more than that in the arms) is very much lower, and may not be recordable. Poor circulation in the legs sometimes leads to delayed healing of injuries or numbness and coldness in the feet.

Evidence of the collateral circulation is often present, and thus is generally most conspicuous in dilated pulsating vessels visible or palpable round the vertebral borders of the scapulae above and below the clavicles, down each border of the sternum and on the abdominal wall. The paths chiefly concerned are by the thoracic axis, the scapular and subscapular anastomoses, and the internal mammary-deep epigastrics but in childhood the collaterals may be quite small, increasing only with time. The diagnosis does not depend upon them, although their presence is conclusive. It is confirmed when notching of the inferior edges of the ribs due to intercostal anastomosis is seen on the chest X ray (X ray 123). If this notching is confined to the right side it again suggests a blockage proximal to the left subclavian artery.

No cardiac murmurs may be heard but usually there is a systolic bruit most audible between the scapulae. A diastolic murmur at the aortic base signifies complicating aortic incompetence a systolic

one with a thrill, aortic stenosis; whereas a machinery murmur indicates the case to be one of the 10 per cent. in which the ductus is patent. The heart may still be normal in size, but often the left ventricle is enlarged; and this is confirmed by the demonstration of left ventricular preponderance on the electrocardiogram. On screening, also, enlargement of the left ventricle is more likely to be seen in older patients. The aortic knuckle is inconspicuous, but the ascending aorta bulges to the right of the sternum and goes farther up the mediastinum than usual.

Aortography is a desirable but not essential step in investigation and is performed by introducing a catheter into a radial artery and passing it up until it reaches the aorta. A contrast medium such as diodone is rapidly injected, radiographs are taken, and an accurate delineation of the character and anatomical position of the coarctation is obtained. In the rare instances which have a long affected segment, or in whom an aneurysm is present, end-to-end anastomosis is out of the question and some form of grafting is required. *The operation should in any case never be undertaken in the absence of suitable grafts*, either homologous grafts preserved by freeze-drying and previously obtained from suitable subjects such as young victims of road accidents, or artificial prostheses of plastic cloth, orlon or vinyon. These must be readily available even if there is reasonable assurance that the coarctation is operable by direct anastomosis. Apart from some element of doubt which remains until the cut ends are actually in apposition, it is always possible that a tear may occur in one or other segment which would prove disastrous in the absence of a graft but can be circumvented if one is to hand. Gross found them necessary in 25 per cent. of patients over the age of 21.

One danger of what is at best a taxing operation is severe bleeding from the friable, tortuous and enormously distended collateral vessels, especially the intercostals, so that any reduction of pressure and blood flow in them contributes to the ease and safety of operating. Thus induced hypotension with intravenous hexamethonium iodide is of value; but care must be taken that when the aortic clamps are finally released a sudden and excessive drop in blood pressure does not occur.

At least six pints of cross-matched blood for transfusion must be available and blood loss throughout is checked by weighing swabs and measuring all blood removed by suction in order that prompt replacement is assured.

Operation

The patient is placed in position for a left posterolateral thoracotomy. It is well for the surgeon to be comfortable and if possible seated with the table tilted towards him. The skin incision is made in short segments at a time to allow bleeding points to be dealt with, and as soon as the deeper layers are reached the individual muscle bellies are freed and divided between clamps so that the larger vessels can be ligated individually and the rest coagulated by diathermy as each clamp is released in turn. The pleural cavity is entered through the bed of the fifth rib which is resected from costo-transverse joint to costal cartilage. If wider exposure yet is needed both the adjacent costo-transverse ligaments are cut. Tortuous and distended col-

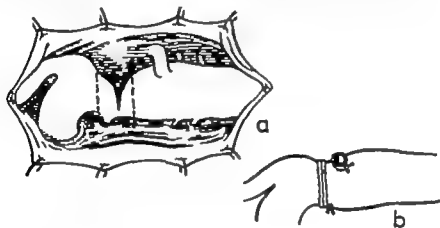


FIG 43 *Coarctation of the aorta*

(a) The coarcted area to be excised, just below the left subclavian and closely related to the ligamentum arteriosum.

(b) After excision the cut ends of the aorta are anastomosed. One large intercostal artery has been tied.

lateral vessels are everywhere apparent, especially in the intercostal spaces and the marked pulsation in the arch of the aorta is strikingly in contrast with the lack of pulsation in the descending portion which is sometimes dilated and thin walled. The coarctation itself is usually just beyond the left subclavian, but can more easily be palpated than seen. Mobilization is begun around the subclavian, and the aortic arch is freed first, as it usually has no intercostal branches. Below the stenosis the dissection of the intercostals calls for great care, for they are sometimes aneurysmal (as well as being very friable) and must on no account be torn at their origins from the aorta, or profuse bleeding which may be very difficult to control will occur.

As a rule two pairs of intercostal arteries are ligated and cut, as well as a bronchial artery arising medially. Tapes are passed round the aorta above and below the coarctation, and traction upon them helps to define the stricture, which is surrounded by much fibrous tissue. The ligamentum arteriosum—or a patent ductus if present—is divided between ligatures; after which the coarcted area becomes much more mobile. Crafoord or Potts clamps are then applied as far away on each side of the constriction as possible, for after it is excised the cut ends tend to retract, and enough room must be left for suturing. A length of about 2 cm usually has to be cut out, but in any case enough must be taken to ensure that the lumen when complete is not less than 60 per cent. of the normal aorta. The ends are then brought together by approximating the clamps, and, providing no undue tension is necessary, they are directly anastomosed (Fig 43). If the gap left after excision of the coarcted segment is too great, an immediate decision must be made to use a graft or prosthesis to bridge it. The exact fate of these is uncertain but many surgeons have now employed them over considerable periods with excellent results.

The anastomosis is carried out with an everting mattress suture of 3/0 silk interrupted and tied at several points. All the knots are left outside and intima is stitched to intima. When the anastomosis is complete the clamps ought not to be removed separately, but both together; and the suture line inspected for leaks. These are controlled by firm pressure, or the insertion of a further stitch or two. The chest is closed with a water-sealed drain in position, after re-inflation of the lung in the ordinary way.

Postoperatively the brachial blood pressure falls, that in the legs promptly rises, and the femoral and popliteal pulses reappear. Episodes of transient but quite severe abdominal pain due to intestinal vascular lesions sometimes follow the increase in pressure. In a few older patients hypertension in the upper part of the body persists, presumably due to long-standing changes in the vessels themselves.

No special postoperative care is necessary, but anticoagulants, which might promote bleeding from the suture line, or needling of the chest, which involves the risk of puncturing one of the large anastomotic vessels, must be avoided.

v. Anomalies of the aortic arch

In foetal life the aortae are paired and give branches to each of the branchial arches, some of which persist as normal adult arteries. Aberrations are not uncommon owing to the survival of trunks which

normally disappear but these are not of importance unless they give rise to symptoms, as they occasionally do. One of the commonest atavisms is for the right subclavian to rise from the left side of the aortic arch and pass across the mediastinum posterior to the oesophagus, which may thus be constricted causing dysphagia. *Dysphagia Lusoria* as it is called, may also be caused by a double aortic arch, one limb of which passes anteriorly, one posteriorly to the mediastinal structures, giving rise sometimes in infants to stridor and cyanosis due to constriction of the trachea, and dysphagia by pressure upon the oesophagus. It may be possible to relieve these by division of the abnormal posterior vessel.

AORTIC STENOSIS, INCOMPETENCE AND ANEURYSMS

STENOSIS AND INCOMPETENCE

AORTIC STENOSIS

AORTIC stenosis occurs as a congenital acyanotic lesion, when the lesion may be at the valve itself or below it in the outflow tract of the left ventricle like the infundibular type of pulmonary stenosis, but it is more often acquired in the course of rheumatic fever. The valve is also affected by atherosclerosis, bacterial endocarditis and syphilis, although in these the principal result is incompetence. It is recognized by the presence of a thrill and a rough systolic murmur in the aortic region, and it is usually accompanied by left ventricular enlargement. Patients often suffer from anginal or syncopal attacks on exertion. Following the striking success of surgery in the treatment of mitral stenosis much pioneer work has been done both in England and America to relieve aortic stenosis in similar ways.

The valve has been approached both from below (i.e., through the myocardium of the left ventricle) and from above by means of an artificial operating tunnel made out of plastic or pericardium sewn onto the aortic wall and used as is the atrial appendage in mitral valvotomy. Unfortunately many factors combine to limit success: the risk of producing incompetence is much greater than in operations upon the mitral valve, and much more serious when it occurs, the commissures are calcified in the great majority of cases, increasing the difficulties and the risk of emboli, and the part of the aorta from which the coronary vessels rise, just beyond the valve cusps, is frequently affected by disease, and cardiac ischaemia is therefore more likely.

Although symptoms tend to appear comparatively late, death follows within one or two years of their onset. Aortic stenosis, like stenosis of any other organ, can be relieved only by surgery, and in view of the inherent technical difficulties, it is important that patients be presented to the surgeon while there is still hope of relieving

them and not when they are *in extremis*, and the heart is already failing.

Brock has recently (1957) reported a series of 130 cases treated by transventricular valvotomy, with a mortality of 10-15 per cent., and good results in 70 per cent. of the survivors. He passes a curved two-bladed expanding dilator, similar to the one employed in pulmonary valvotomy, through the wall of the left ventricle, and opens it in the aortic valve after the carotids have been temporarily occluded to avoid risk of cerebral calcific emboli.

Although this method has proved so successful, it is probable that direct approach to the valve under hypothermia will replace it. The circulation is interrupted by closing the venae cavae (as described on p 302) the aorta clamped distally, its wall opened above the valve which is directly inspected and its commissures freed, if necessary with scissors. The incision in the aorta is then clamped, the circulation resumed, and the aortic wall finally sutured.

AORTIC INCOMPETENCE

The only operation that has had any success in this disorder is that of Hufnagel of Washington who divides the descending aorta and interposes into it a plastic ball valve. Half of his 80 patients were already in a terminal state, and the rest very ill. Only 5 died during the operation and the hospital mortality was 20 per cent. In the rest the operation was successful, the patients becoming used to the continual rattle of the ball valve which was more audible when they stood with their mouths open. Twenty per cent. however died within 2 years and it is still too soon to know the ultimate fate of this prosthesis.

AORTIC ANEURYSMS

Tuffier, who first demonstrated in 1902 that most syphilitic aneurysms have a narrow mouth which can be closed laid down three requirements for their successful removal that they should be accessible, isolable and extricable. Few conformed to these essentials at the beginning of the century few do not today.

The principal causes of aneurysms are *syphilis arteriosclerosis* and *trauma*. Most of those due to the first are saccular representing quite localized blow-outs of the wall, with the rest of the aorta comparatively sound though inevitably affected by syphilis and lend

themselves to excision. Most are seen in patients under the age of 55; are situated in the intrathoracic portion of the aorta, are associated with bone erosion and severe pain; and are likely to cause death within 6-9 months.

In contrast, most due to arteriosclerosis are in patients over 60, are fusiform, are not associated with severe pain or bone erosion; are generally abdominal in position, and on the whole have a better prognosis.

Traumatic aneurysms are chiefly seen in victims of traffic accidents, and are caused by sudden shearing forces which partially tear the aortic wall giving rise to an aneurysm. The commonest site for them is at the beginning of the descending aorta just below the left subclavian; and they are likely to be very localized, as more extensive damage is incompatible with survival.

Congenital and mycotic aneurysms also occur, the former sometimes with coarctation, but are rare (X-ray 123).

Many intrathoracic aneurysms have now successfully been excised, the difficulties met with being chiefly due to the necessity for maintaining a continued outflow from the heart, and a continued cerebral circulation. Those of the *ascending aorta* (a common site for syphilitic aneurysms) can only be dealt with if they have a sufficiently narrow mouth that can be clamped and closed. Aneurysms of the *arch* itself (X-ray 124) or of the *innominate artery*—the second commonest place for intrathoracic aneurysms—may need the insertion of a by-pass graft. This is first sewn on to the side of the ascending aorta, a section of whose wall is occluded by curved finely-toothed clamps, and to the healthy vessel beyond the aneurysm, which may then be dealt with providing one carotid is left intact. When the aneurysm is in the *descending aorta* the whole vessel is clamped, as during an operation for coarctation, and the aneurysm either opened and its neck sutured, or bodily excised, and continuity re-established by a homo-graft or a plastic prosthesis.

The situation and dimensions of aneurysms can sometimes be determined preoperatively by aortography with an opaque medium; and surgical exposure varies with their type and position, but in all cases must be liberal. The operation of wrapping aneurysms with cellophane or of inserting wires into them should only be considered if they are irremovable.

them, and not when they are *in extremis*, and the heart is already failing.

Brock has recently (1957) reported a series of 130 cases treated by transventricular valvotomy with a mortality of 10-15 per cent., and good results in 70 per cent of the survivors. He passes a curved two-bladed expanding dilator, similar to the one employed in pulmonary valvotomy, through the wall of the left ventricle, and opens it in the aortic valve after the carotids have been temporarily occluded to avoid risk of cerebral calcific emboli.

Although this method has proved so successful it is probable that direct approach to the valve under hypothermia will replace it. The circulation is interrupted by closing the venae cavae (as described on p 302) the aorta clamped distally its wall opened above the valve, which is directly inspected and its commissures freed if necessary with scissors. The incision in the aorta is then clamped the circulation resumed, and the aortic wall finally sutured.

AORTIC INCOMPETENCE

The only operation that has had any success in this disorder is that of Hufnagel of Washington who divides the descending aorta and interposes into it a plastic ball valve. Half of his 80 patients were already in a terminal state, and the rest very ill. Only 5 died during the operation and the hospital mortality was 20 per cent. In the rest the operation was successful the patients becoming used to the continual rattle of the ball valve which was more audible when they stood with their mouths open. Twenty per cent. however died within 2 years, and it is still too soon to know the ultimate fate of this prosthesis.

AORTIC ANEURYSMS

Tuffier who first demonstrated in 1902 that most syphilitic aneurysms have a narrow mouth which can be closed laid down three requirements for their successful removal that they should be accessible, isolable and extricable. Few conformed to these essentials at the beginning of the century, few do not today.

The principal causes of aneurysms are *syphilis*, *arteriosclerosis* and *trauma*. Most of those due to the first are saccular representing quite localized blow-outs of the wall with the rest of the aorta comparatively sound though inevitably affected by syphilis and lend

themselves to excision. Most are seen in patients under the age of 55; are situated in the intrathoracic portion of the aorta, are associated with bone erosion and severe pain; and are likely to cause death within 6-9 months.

In contrast, most due to arteriosclerosis are in patients over 60; are fusiform, are not associated with severe pain or bone erosion; are generally abdominal in position; and on the whole have a better prognosis.

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THE SURGERY OF THE PERICARDIUM

PERICARDIAL EFFUSION AND TAMPONADE

CARDIAC compression may occur due to haemorrhage or to effusion into the pericardial sac and may be acute (as in the former) or chronic and insidious in onset as is seen in chronic constrictive pericarditis. However it is caused, by wound or by inflammation, cardiac tamponade (as it is called) results in (1) a quiet heart because the apex beat and the sounds are muffled by the fluid, (2) a falling arterial pressure because the left ventricle finds it progressively more difficult to pump out blood and (3) a rising venous pressure because the heart is unable to accept the blood returning to it. As a result the patient is faint and pallid, the pulse is poor and rapid, and after a time the neck veins become engorged. Such symptoms call for urgent relief. The heart shadow may be enlarged if the pericardial sac is extensible, but in acute cases this has not time to happen and the heart appears of normal size.

If tamponade is due to a stab wound or to postoperative haemorrhage there is clearly no time to be lost in opening the chest, sewing up the wound or securing the bleeding vessel and clearing the clot out of the pericardium but if it is of more gradual onset due to effusion (which may be postoperative or inflammatory and sometimes purulent) relief can be given and the diagnosis confirmed by pericardial aspiration.

This should be performed with the patient propped up in bed with his back at an angle of about 45°. After the skin has been cleaned and local anaesthetic injected, a fairly wide bored needle attached to a Martin's syringe with two-way tap is passed in at the angle formed by the xiphisternum and the left costal margin, and directed steadily upwards and backwards until it enters the diaphragmatic surface of the pericardium. Here the risk of pricking a coronary vessel or the heart itself is least. Whereas the latter accident might have no ill effects the former would probably be fatal. The effusion can then be safely aspirated, though care must be taken towards the end as the heart comes closer and its pulsation is felt. If the fluid is turbid it is sent for culture and penicillin or streptomycin solution instilled into the sac according to the aetiology. Frankly purulent collections

call for open drainage of the sac through the bed of the resected seventh left costal cartilage.

CONSTRUCTIVE PERICARDITIS OR PICK'S DISEASE

A boy of 12, whose father was being treated in another hospital for pulmonary tuberculosis, was admitted complaining of breathlessness and palpitation. He had been unable to play games and felt ill and weak. On examination he was found to have a low fever, and a small right pleural effusion. His apex beat was hard to feel and the cardiac dullness and outline much increased. There were no murmurs; and his lung fields were quite clear except for the effusion. He was kept strictly in bed for several months during which the temperature became normal and the effusion disappeared completely. An attempt was made to aspirate the pericardial effusion which was thought to be present, but it was not very successful, and only a little serous fluid was obtained which proved sterile on culture. A guinea pig was inoculated with some of it, and was found to be infected with tuberculosis when it was killed six weeks later. Streptomycin and para-aminosalicylic acid were prescribed, and the size of the pericardial sac became distinctly smaller. As it did so the boy's urinary output dropped, his external jugular veins became distended, and his liver edge descended below the right costal margin. He felt better in himself and was not breathless as he lay in bed, but the least exertion made him pant and greatly increased his pulse rate. His liver increased in size and he developed ascites, which soon made his abdomen look like the belly of a frog. There was, however, no swelling of his ankles at this time, though pitting oedema appeared later.

After further treatment and investigation he was submitted to surgery for cardiac decompression. When his left chest was opened no heart beat could be seen at all except for pulsation in the great vessels. The pericardium was enormously thickened, but after its parietal layer had been removed there was still no movement of the ventricles visible. The pericardial sac itself was virtually obliterated, containing only some thin fluid and caseous material; and the visceral layer of pericardium was reddened, irregular, and very thick. It was incised down to the heart muscle and peeled off in postage-stamp fragments. As this was done the myocardium bulged through the gap, beating strongly, like a living creature trying to force its way out of a strait-jacket. Both ventricles were completely freed. Within a few days the urinary output rose sharply and the neck veins emptied. The ascites disappeared and the liver slowly receded. The boy is now a keen footballer.

This case history exemplifies very well the evolution of constriction following on the absorption of an infected effusion due to tuberculous pericarditis. Other forms of pericarditis—such as that due to rheumatism—cause pericardial adhesions but they do not lead to cardiac compression, although in a few rare instances of suppurative pericarditis due to a staphylococcal infection, this may happen. For all practical purposes *constrictive pericarditis is a direct consequence of tuberculous pericarditis*. In early and acute cases (such as that described) it may be possible to demonstrate tubercle bacilli, but in later ones and especially those that have calcified, it is not. Occasionally a tuberculous lesion is present in the lungs, or there is a significant history of contact, whereas in others the evolution of the disease can be watched from the first onset of tuberculous pericarditis to its final contracture into cardiac constriction. More often positive confirmation of the tuberculous aetiology is wanting, and the patient is in any case not seen until long after his initial pericarditis has burnt out, his symptoms being due solely to the strangling rigidity of fibrosis and calcification.

As the effusion which accompanies a tuberculous pericarditis slowly absorbs, leaving only a caseous deposit between the inflamed layers of the pericardium, the sac itself is obliterated, or nearly so and the parietal and visceral layers become densely adherent to each other and to the heart. As happens during all tuberculous healing, there is intense fibrosis and thickening of both pericardial layers, with gradual contraction and compression of the heart within. Not only are the ventricles unable to contract freely and the heart to shorten on its long axis (as it must do in systole) but it is also unable to expand, and to lengthen in diastole. Venous blood cannot be received back into the right ventricle but is forced as it were to queue up in the caval veins awaiting admission, thus leading to a rise in venous pressure and distension of the neck veins. The heart's intake and output are both severely and arbitrarily limited and they cannot be increased in response to effort. As a result, although the patient is not breathless at rest, and can lie quite flat in bed without discomfort (as he could not do were he in congestive failure) he at once becomes dyspnoeic on the least exertion. The output per beat cannot be increased, and exertion is therefore attended by a disproportionate rise in the pulse rate. The pulse itself is of poor volume and may be paradoxical—that is decreasing on inspiration. The systolic blood pressure and the pulse pressure are low the former often being 90 or 100, and the latter 15 to 20 mm. of mercury instead of the usual 30–40 mm. The velocity of the blood flow—

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measured by injecting a substance such as saccharine into an arm vein and timing the appearance of its taste in the mouth—is likewise decreased, the normal arm-to-tongue time of 16 to 20 seconds being increased to 40 or even 60 seconds. The blood volume on the other hand is increased by 30 or 40 per cent.

Because of the diminished intake of blood the venous return is progressively dammed up so that the rising venous pressure causes distension of the neck veins and enlargement of the liver. The external jugulars are not distended when a normal person reclines at 45°; but as the venous pressure rises they fill, sometimes up to the angle of the jaw. The fact that they also pulsate with the heart beat proves that mediastinal obstruction, such as is caused by growth or a goitre, is not responsible for their state. Catheterization shows the pressure in them often to be as high as 15 or 20 mm. of mercury. Venous stasis may lead to coldness of the extremities and peripheral cyanosis.

Hepatic enlargement and ascites tend both to be earlier in the syndrome and much more severe than is the case in congestive heart failure, and oedema of the legs and ankles is less marked. This may partly be due to the absence of valves in the hepatic veins, and partly due to their orifices being held rigidly open by fibrosis. It seems likely that the pleural effusions which occur may be inflammatory exudates rather than passive transudates, for they are usually small, transitory and unilateral; but the ascitic fluid is unlikely to be so for it disappears promptly after operation.

As the action of the right ventricle is also impeded, blood flow to the lungs is reduced. Thus no moist sounds are heard in them and they look clear radiologically—a striking contrast to their state in congestive heart failure.

The heart itself is classically small and quiet, but sometimes it is rather bigger than usual due to accumulated debris in the thickened pericardial sac. The apex beat is weak or imperceptible; and there are no murmurs, although a third heart sound can often be detected after the normal second sound, which is distant and subdued. Too much significance ought not to be attached to the prominence or otherwise of the heart beat and its sounds, for there is considerable variation in this respect depending on the distribution and extent of constricting fibrosis or calcification.

Atrial fibrillation is present in a quarter of all cases, and is associated with some degree of myocardial atrophy due to splintage and disuse in patients whose disease is long-standing and whose diagnosis has been tardy. In most instances pathological changes are confined to the pericardial layers, and the heart itself is otherwise quite normal.

This fact, and the additional risk of permanent liver damage are reasons why surgical relief should not be delayed too long.

The electrocardiogram is consistently of low voltage in all leads, and the T waves are flattened or inverted, the degree of inversion providing some measure of the myocardial involvement. The blood serum-protein ratio is often reversed, but this does not seem to be related directly to liver damage being rather the consequence of the accompanying fluid and salt retention.

Radiologically the cardiac outline is small, but its most striking feature is its lack of pulsation. The degree of this relative immobility varies from different aspects, tending to be most marked over the apex and left ventricle.

In long-standing cases calcification is common—as it is in all examples of healed tuberculosis—and may completely ensheath the heart, or lie mostly on the dependent diaphragmatic surface of the pericardium like a cup (X rays 125-126). Its presence greatly simplifies diagnosis and it is found in a quarter to a third of all cases. Tomography sometimes reveals its presence when this is not evident on plain films.

The conditions most likely to be confused with constrictive pericarditis are congestive heart failure, cirrhosis of the liver, and, perhaps, polyserositis. The bedside contrasts it provides with the first of these—the absence of pulmonary engorgement, or of cardiac enlargement, or of murmurs; the disproportionate hepatic enlargement and ascites, the ability to lie flat without dyspnoea—are all sufficiently striking. In cirrhosis, one of the cardinal signs of constrictive pericarditis, a raised cervical venous pressure, is wanting. If the three features, a small quiet heart, raised venous pressure and ascites are borne in mind mistakes in diagnosis are unlikely.

The optimum time for surgery which alone provides a cure depends upon the stage the disease has reached when it is first seen. If active tuberculosis is thought still to exist, or has not long been quiescent, a period of rest and observation is necessary together with a full course of streptomycin and para-aminosalicylic acid. 1 G. of streptomycin (with 5 G. four hourly of para-aminosalicylic acid) should be given daily for one month preoperatively unless the pericardium is calcified. Paracentesis abdominis is often required. The fluid intake is limited to 1,500 cc. and salt to about 2 G. a day. If plasma proteins are low a high-protein diet is indicated and mercurial diuretics (such as mersalyl) are given every third or fourth day to help excretion. A careful fluid intake-output chart is always kept, and the increased output which follows operation provides

some measure of its success. Digitalis is usually contra-indicated because it tends in these cases to diminish cardiac output still further, but when fibrillation is a serious factor it may be essential. Secondary anaemia requires correction but blood transfusion is best avoided if possible. When increasing cardiac compression is evident, operation should not be delayed even in the face of recent tuberculous activity nor should it be so long postponed in older patients that permanent myocardial or hepatic damage is allowed to occur.

Pericardectomy

The object of the operation is to free both ventricles as completely as possible from their corset of constricting fibrous or calcified pericardium. Although some difference of opinion exists on the subject I am quite satisfied that it is wholly unnecessary as well as dangerous to make any attempt to clear the atria or the great vessels. It has been shown on many occasions, both practically and experimentally, that the caval veins can be narrowed much more than ever occurs in constrictive pericarditis without ill effect. Failure or unsatisfactory results following pericardectomy are due to either insufficient liberation of the ventricles, in particular the apex; or, in late cases, to secondary myocardial atrophy. Removal of the thickened pericardium should be as complete as possible over the ventricles; and it should stop at the atrio-ventricular groove.

The heart is approached by an antero-lateral incision, with subperiosteal resection of the fifth left rib and its cartilage up to the sternal edge. The pleura is always densely adherent to the pericardium and no attempt is made to operate extrapleurally. After the lung is freed it is packed away from the heart, and the phrenic nerve is identified as it runs down on the pericardium. Every care is taken thereafter not to injure the nerve.

The parietal pericardium is incised with a knife until a plane of cleavage between it and the visceral layer is found, representing what is left of the pericardial cavity. Sometimes this contains caseous material, and sometimes it is hardly identifiable. It is developed until a flap of pericardium can be raised on both sides and used thereafter for retraction. The true constricting coat, which it is imperative to remove, is that formed by the visceral pericardium which must be incised down to the heart muscle itself. This plane of cleavage is usually much less easy to develop than the first and dissection is carried on to the surface of the left ventricle, which must be freed before the right lest pulmonary congestion ensue. The fibrous sheath, or calcified plaques, are lifted little by little, in postage-stamp areas,

the myocardium bulging through with each extension of the gap. Every precaution is taken to avoid damage to the left coronary artery running down the interventricular sulcus, and if it is closely invested in fibrous tissue a strip should be left and the dissection begun again on its other side. The apex is the most important area of all to free, for unless this is done the shortening of the heart in its long axis such an important part of systole, cannot take place.

Finally, attention is given to the right ventricle, which is similarly freed, dissection always stopping at the A.V. groove. When clearance is complete all bleeding from the cut pericardial edges is stopped, the lung re-inflated, and the chest closed, with a water-sealed drain. The only serious risk of the operation, apart from coronary damage, is accidental rupture of the heart. This is more likely to occur if the atria are trespassed on, but may happen if the plane of dissection is lost, especially over the right ventricle. Such haemorrhage is controlled by digital pressure until a few deep sutures can be passed through the myocardium and a flap of pericardium sewn down over the hole.

The first sign of recovery is usually a sharp rise in urinary output, but this is often delayed for several days, or even some weeks during which time the patient remains in bed. Once it begins subjective improvement is rapid, but it may take many weeks for the ascites to disappear, the liver edge to recede, and for the high venous pressure to subside. Failure to improve is usually due to inadequate decortication of the ventricles and calls for further operation.

MITRAL STENOSIS AND ITS SURGICAL TREATMENT

A QUARTER of all cardiac invalidism stems from rheumatic heart disease; and in more than half of these cases (constituting at least a quarter of a million people in Great Britain alone) mitral stenosis is the predominating lesion. This contrasts with only $2\frac{1}{2}$ per cent. of cardiac patients having congenital defects such as Fallot's tetralogy. The virtual conquest of mitral stenosis in recent years not only constituted a major surgical advance, but opened the way for other bolder proceedings, and proved that the heart tolerates intervention no less well than other organs. Cardiac surgery has the same objects as surgery elsewhere: the restoration of normal anatomy; the relief of obstruction; and the preservation of blood supply.

Co-operation must exist between surgeon and physician, for it is upon the selection of cases as well as upon efficient technique that success in mitral valvotomy depends. The surgeon must spend much of his time in an efficient cardiological department with the most modern diagnostic facilities, just as the cardiac physician must visit the operating theatres where his diagnosis and selection receive their ultimate test.

A stricture at the mitral valve calls for relief just as does one in the bowel, the bronchus, or the urethra. One must first be quite sure, however, that the patient's symptoms stem from stenosis and not from regurgitation, from other valvular defects, or from myocardial degeneration. Furthermore some mitral valves have been so severely damaged by disease that surgery can never restore their anatomy or their function. In others various adverse factors militate against a good result but do not in themselves exclude it. Finally it is not justifiable to subject to operation patients who have the physical signs of mitral stenosis but who suffer no disability from it. The results of surgery are excellent in 60 per cent. of patients; another 20 per cent. are improved, some considerably so; and the rest are left as they were, or worse. The average operative mortality is less than 5 per cent.

Mitral stenosis is four times commoner in women than in men; and in nearly half the cases there is no clear history of acute rheumatism. Symptoms may first appear at any age, but are commonest

in the early thirties, and the average period of survival from their commencement is 18 years, the average age at death being 47

Symptoms

The first and principal symptom is shortness of breath, at first hardly noticed except on running or unusual exertion, and the importance attached to it varies widely with the temperament of the patient. A man should be asked if he can do similar work, and keep up with men of his own age. A woman whether she can do her house work, how many stairs can she climb without halting? How far can she walk or run? Disability is at first slight, but soon the patient is unable to do the heavier chores or walk very far without distress later still is made breathless even by the lightest tasks, is easily fatigued, and can no longer lie flat in bed without discomfort and finally reaches a stage of total invalidism.

Many patients first complain of coldness in their hands and feet, and these may be cyanosed as a result of vasoconstriction and stagnation. Tightness across the chest, the inability to take a full breath, and even retrosternal pain which must be distinguished from angina are common. Others suffer from bronchitis and winter coughs some due to congestion of the lungs, some to intercurrent infections. Isolated haemoptysis, occasionally profuse is often seen at an early stage, when it is not necessarily of serious import, unless it is due to embolism. Smaller, repeated bleedings occurring later, are more serious, for they indicate increasing pulmonary venous congestion. Haemoptysis is especially common in pregnancy at the conclusion of which it may cease. The same is true of the paroxysmal attacks of breathlessness which often accompany gravidity and denote a patient poised on the knife-edge of pulmonary oedema. Association with toxæmia in pregnancy is particularly dangerous. It should be noted that mitral stenosis is no longer an indication for ending pregnancy *it is an indication for valvotomy*

Dyspnoeic attacks of this type are not confined to pregnant women, although pregnancy additionally burdens an already inadequate heart. They occur in many patients, and are associated with sudden increases of pulmonary venous congestion due to obstruction to the blood flow through the mitral valve (X ray 128). When pressure in the capillaries concerned equals the pressure in the cells at which osmosis takes place, pulmonary oedema floods the lungs. Such alarming attacks are sometimes precipitated by emotion or by unusual exertion as well as by pregnancy. They constitute a strong indication for valvotomy. Many patients are orthopnoeic, able to

sleep only if they are propped up with pillows, and waking with a sense of suffocation should they slip down from them. The number of pillows needed for comfort provides a rough indication of the severity of the condition.

Many patients have suffered one or more episodes of right heart failure, with enlargement of the liver or peripheral oedema. In some, and with time, the pulmonary vessels respond to the increasing tension in them by vasoconstriction of the arterioles, helping to protect the lung against congestion, and preventing the paroxysmal attacks of breathlessness. But this throws the burden back instead upon the right ventricle, causing first its hypertrophy and ultimately its failure—a consequence which can be averted only by surgery. The onset of right heart failure when the heart is in normal rhythm is of grave prognostic significance, and 60 per cent of such patients are dead within a year.

The incidence of atrial fibrillation increases steadily with age, being present in 40 per cent of patients submitted for operation. In some the attacks are intermittent, but almost invariably they precede permanent irregularity. This is rarely reversed after valvotomy.

Emboic phenomena are unusual if the heart is in normal rhythm, but common when it is fibrillating. Cerebral embolism is so grave a catastrophe that the need for surgical prevention, and the avoidance of recurrence, outweighs any other unfavourable factors.

Examination

Patients with fully developed mitral stenosis tend to be pinched and thin, with cold hands and feet and noses, and a malar flush. The pulse is small in volume due to the left ventricle's diminished output. The apex beat has a slapping quality—the blow of an open hand, contrasted with the heavier clenched fist of left ventricular hypertrophy. If there is much right ventricular hypertrophy a systolic lift may be evident down the left border of the sternum. A diastolic thrill is often felt. The first heart sound at the apex is characteristically loud and snapping, and should this not be so it raises doubts as to stenosis being the predominant lesion. Particular loudness and crispness is a favourable sign, and suggests pliant valve cusps coming smartly together. The classical rumbling presystolic, and mid-diastolic, murmurs of mitral stenosis are present unless there is fibrillation also, when the presystolic murmur will not be heard. An absent or faint diastolic murmur suggests much hypertension or a very tight stenosis.

Midway between the apex and tricuspid area the 'opening snap, coming just after the second heart sound, should be audible, and when well marked and clear is confirmatory evidence of predominant stenosis and pliant valve cusps. Absence or diminution of this sound suggests either that the cusps are calcified, or that a good deal of incompetence is present. Increase of the pulmonary second sound is generally indicative of pulmonary hypertension although its quality varies much in the same individual with exercise or emotion. It is normally split, but if the splitting is very close, or absent altogether the pulmonary pressure is probably high.

Radiological screening is an indispensable part of examination. In general the lung fields show some congestion, with increased vascular markings and, occasionally evidence of infarction or of haemosiderosis. The heart at first appears generally enlarged and it is important to distinguish if possible between increase in size of the right ventricle and of the left, left ventricular enlargement indicating predominant regurgitation which contra-indicates surgery (This distinction is sometimes very difficult to make by screening alone.) The more normal the size of the left ventricle the better are the surgical prospects. The aortic knuckle is inconspicuous, either because it is actually smaller than usual (if the disease has been present from childhood) or because it is overshadowed by enlargement of the pulmonary artery just below it—a fairly constant finding which reflects the degree of hypertension present. Below this again is the prominence made by the left atrial appendage the absence or diminution of which provides a warning of possible difficulty in gaining surgical access to the atrium. The left cardiac profile ends in the normally sized apex of the left ventricle. Both the left atrium and the right ventricle may be enlarged to varying degrees, and they are studied best in the right and left anterior oblique positions respectively. In particular it is necessary to look carefully for systolic expansion of the left atrium (especially if it is very large) for this indicates marked regurgitation. Calcification of the mitral cusps is sometimes seen on direct screening but is more likely to be revealed by tomography.

Right ventricular preponderance on the electrocardiogram is an indication for valvotomy. Left ventricular preponderance is a contra-indication for it means regurgitation or aortic disease is predominant.

Cardiac catheterization is unnecessary. If any likelihood of active rheumatism exists the erythrocyte sedimentation rate must be checked.

Selection of cases

The first and most crucial point to be decided about a patient with mitral stenosis is the extent of regurgitation present: for all stenotic valves permit *some*; and valvotomy often, by restoring a greater measure of normal function, diminishes its degree. In the same way many patients have a small aortic leak. When incompetence is the *predominant* lesion, however, surgery can do no good, and frequently does harm by increasing the leakage. The presence of an apical systolic murmur, with or without a thrill, although highly suspicious, is not conclusive. The regurgitant murmur is blowing in quality, and the louder it is the worse the leak is likely to be. The absence of such a murmur does not exclude reflux, and in any case confirmatory evidence must be sought: a powerful left ventricular thrust at the apex; the absence or reduction in intensity of the first heart sound, or of the opening snap, when there is no calcification of the valve; the absence of a presystolic murmur when the heart is not fibrillating; the presence of systolic pulsation in a big left atrium, and finally left ventricular preponderance on the electrocardiogram, *all indicate mitral incompetence and therefore contra-indicate surgery*. An aortic diastolic murmur conducted down the sternum and a muffled second sound must always be sought with the possibility of an aortic leak in mind. A small leak must often be accepted, but one in which the left ventricle is enlarged is also a contra-indication.

Operation is obviously undesirable in the presence of active rheumatism, so it is not often advised under the age of 20 or 25; but if symptoms of obstruction are increasing it may have to be considered. In the same way long-standing valvular disease is likely to be associated with myocardial degeneration in elderly patients with the result that the heart is unable to respond satisfactorily to mechanical relief of the valve, and the postoperative course is disappointing. For this reason patients much over the age of 50 are usually rejected unless there are pressing indications for surgery. Atrial fibrillation implies some degree of myocardial damage and is usually irreversible; it is therefore an unfavourable element but not a contra-indication. Neither is calcification of the cusps, although it may increase the difficulty in separating them and there is an added risk of embolic fragments being dislodged by the finger as it crumbles the hard nodules on the cusp edge. In fact, however, I have often found valves with much calcification still to be diaphragmatic in shape, to separate quite easily, and to give excellent results, whereas some of the worst valves, with extreme deformity and much scarring, are devoid of calcium and yet are virtually inoperable.

Indications for early surgery are steadily increasing breathlessness, paroxysmal pulmonary oedema, frequent haemoptysis or right ventricular preponderance. Urgent indications are emboli, pregnancy and attacks of right heart failure. Time should always be devoted to preparing patients for surgery. It is perhaps only in pregnancy that mitral valvotomy becomes an emergency operation, and it should always be performed, if possible, before the sixth month.

Contra-indications to valvotomy are the absence of disability, the presence of active rheumatism, age over 50 and signs (such as left ventricular preponderance *vide supra*) that mitral or aortic incompetence is the principal lesion.

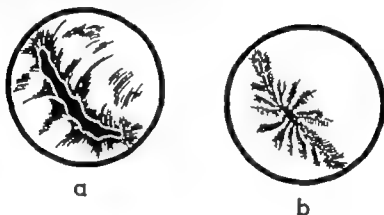


FIG 44

View of normal and stenosed mitral valves from the left atrium.

(a) The direction of the valve orifice is seen between the larger antero-medial cusp and the smaller postero-lateral one.

(b) In stenosis the anterior and posterior commissures are fused, leaving only the stenotic orifice between them.

The best results are to be looked for in patients between the ages of 25 and 40 with normal rhythm and small hearts, in which the only detectable lesion is mitral stenosis, and the valve cusps retain their supple and membranous quality. Favourable physical signs are therefore a small volume pulse and a slapping apex beat, a normal sized left ventricle, a loud sharp first heart sound at the apex, and a crisp clear 'opening' snap.

The mitral valve consists of two membranous cusps: a larger and more important antero-medial, or septal, one on the side nearer the aorta, and a smaller postero-lateral cusp on the outer side. They come together (and fuse together when inflamed) along a line which runs obliquely from about 10 o'clock in front to 5 o'clock behind, as the valve is viewed from the atrium (see Fig 44). During diastole

the cusps normally fall apart to allow the ventricle to fill, but as the ventricle contracts their edges snap together and their membranous surfaces, especially that of the large antero-medial cusp, belly up into the atrium like a spinnaker full of wind. They are held in check, and their edges precisely aligned, by the chordae tendineae inserted fan-wise along them, and tethered below to the papillary muscles which rise like stalagmites from the ventricular floor. The thickest and strongest of these chordae are attached to the cusp edges at about the junction of the middle and outer thirds (described by Brock as 'the point of critical insertion') and it is probably here that fusion of the edges first occurs, spreading outwards to the valve ring on the heart wall, and slowly inwards to the centre, until only a rigid hole of 0.5 to 1 cm. is left through which blood can pass. The rheumatic process attacks not only the cusps, but their chordae, which are an integral and important part of the valve mechanism. In milder cases the chordae may not be seriously affected, but remain long, so that the cusps are able to retain their normal position as a horizontal diaphragm between atrium and ventricle, and after separation of the commissures are able to function as normal valves again, even if the commissures were calcified. When the chordae are severely scarred they become shortened, thickened, and matted together, and the cusps are drawn down into the ventricle as a sclerotic cone at whose apex is the orifice, and whose walls are no longer at right angles to the walls of the ventricle, but parallel, and closely contiguous with them (Fig 45). This relationship may be difficult to feel with the finger if it is not understood, and it is of great importance not merely to *dilate* the central orifice, but to *split* the lines of fusion right out to the valve ring, and to separate if possible the matted chordae below the valve. Only in such a way can the cusps be made mobile again. Rigidity of the orifice makes some regurgitation inevitable, and if contraction of the chordae is uneven this regurgitant jet will be directed sideways against the atrial wall instead of directly upwards. If mobility can be improved the degree of regurgitation is sometimes much diminished.

When cicatrization is very severe the chordae are altogether destroyed, and the deformed cusps fused to the papillary muscles. It is easy to see that in such a case even the more complete separation of the commissures cannot be expected to restore function to the valve; and indeed the condition is virtually inoperable.

Although a guess can be made preoperatively, from the physical signs and the severity of symptoms, at the degree of valve deformity present, it remains a guess; and it is only when the valve is directly

palpated with a finger that an accurate diagnosis and prognosis can be made

Preoperatively as much time as necessary is devoted to getting the patient into the best possible state for surgery. In those who are fibrillating no attempt is made to restore normal rhythm with quinine but nearly all, whether in sinus rhythm or not, are digitalized

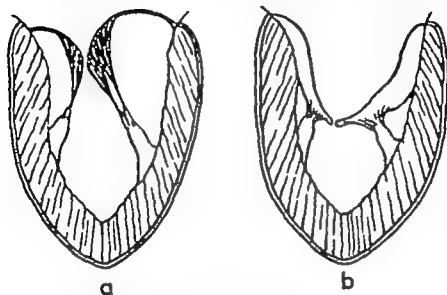


FIG 45

(a) Normal mitral valve cusps with their chordae tendineae.

(b) The effect of severe rheumatic endocarditis. The cusps are enormously thickened and retracted so that they lie almost parallel with the ventricular walls. The chordae are destroyed, and the cusps almost fused with the papillary muscles. Stenosis of this type is inoperable. Between the two extremes lies every gradation of deformity

Should any degree of heart failure exist, medical treatment by rest, restricted fluids, mercurial diuretics, and limitation of salt intake is continued so long as it produces improvement. Anaemia if present is corrected, and a few days before operation penicillin is begun and is continued subsequently for a week or ten days. It is the duty of one of the surgical assistants to feel the pulses in all four limbs, and to check them again immediately after operation.

Mitral valvotomy

The patient lies in the usual position for a left postero-lateral thoracotomy but without a chest rest and, after infiltration with local anaesthetic, the chest is entered through the bed of the fifth rib which

is not resected. The lung is palpated, an estimate made of the degree of induration found in it, and if desired a biopsy taken from its lingula process. The lung is displaced posteriorly, covered with a wet warm towel, and held in place by a malleable lung retractor; the pericardium is thus exposed. The operating table is now tipped towards the surgeon.

The pericardium is picked up and opened vertically just behind the phrenic nerve to give wide access to the side of the left atrium and its appendage. It is most important that the phrenic nerve should suffer no injury or handling at any time during the operation. A stay suture is inserted at each end of the anterior pericardial flap, clipped, and allowed to hang down away from the surgeon in order to retract the flap. No local anaesthesia is used in the sac, and irritability of the heart (as evidenced by extra systoles) is seen most often in patients insufficiently digitalized preoperatively. It is doubtful if the addition of procaine to the intravenous drip is of much value.

The heart is thoroughly examined, and the rhythm and force of its beat, the presence of systolic expansion of the atrium, and the size of the pulmonary artery noted. At the apex a characteristic diastolic thrill is felt; whereas a systolic thrill over the atrium suggests significant regurgitation. With his left thumb in the transverse sinus, and his index finger on the front of the aorta, the surgeon palpates the region of the aortic valve and forms an estimate of its condition by noting any thrill or post-stenotic dilatation. If necessary direct pressure recordings are taken from it and from the left ventricle.

The atrial appendage affords the easiest access to the mitral valve (Fig. 46a). It varies greatly in size and shape and may be too small and narrow for use, or even vestigial. In the great majority of cases, however, it is of suitable dimensions, and is occasionally aneurysmal. It must be most carefully felt for the presence of clot, and should any be suspected its wall is incised directly so that the pressure of blood within expels the clot if it is loose. If the whole appendage is full of clot it cannot safely be employed, and access to the valve must be gained through the atrial wall by the use of a Rumel snare. Providing it is clear, however, or the clot is confined to its tip, a fabric-covered curved clamp is applied to its base, care being taken to avoid the coronary vessels lying medially. No purse-string of any kind is necessary, or should be used.

An incision just long enough to accommodate the surgeon's index finger is then made at a convenient point on the lateral wall of the appendage and another stay suture is inserted at each end of the upper leaf to give the assistant control during application and

removal of the clamp (Fig. 46b) The interior is inspected for clot, any present evacuated, and the clamp removed for a moment to allow a jet of blood from the atrium to wash chance fragments from the interior If the atrium itself contains clot that cannot be washed out, the situation is a dangerous one and the safest approach to the valve is probably via the lumen of the superior pulmonary vein, a clamp

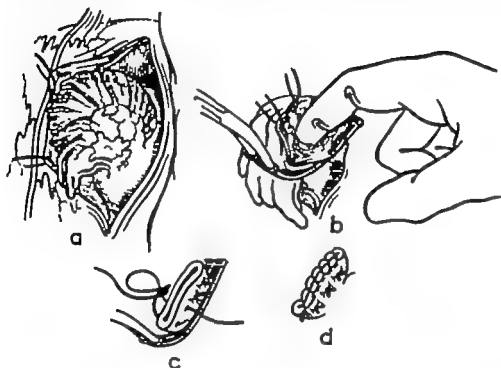


FIG 46. *Mitral valvotomy*

- (a) The pericardium is opened to give access to the atrial appendage.
 (b) The appendage is opened after a light clamp has been applied to its base, and the index finger introduced.
 (c and d) After valvotomy the appendage is amputated and its base closed by interrupted mattress sutures and a final continuous layer

being applied where the vein enters the atrium, and its branches from the lung controlled by tapes.

If the appendage is too small to admit the index, it may still be possible to perform the operation with the little finger but if neither can be introduced, or the appendage is wanting altogether or obstructed by firm organized clot, the approach to the valve must be made directly through the atrial wall. An oval purse string, long enough to enclose the index finger is made on the atrial wall with No 40 linen thread between the base of the appendage and the

superior pulmonary vein (Fig. 47). The ends of the suture are left long and threaded up the flexible shaft of a Rumel snare which is held on the side away from the surgeon, by his assistant. The myocardium inside the purse-string loop is incised down to, but not through, the endocardium, the final rupture being made by the finger itself. As the index finger enters the atrium the snare is pulled

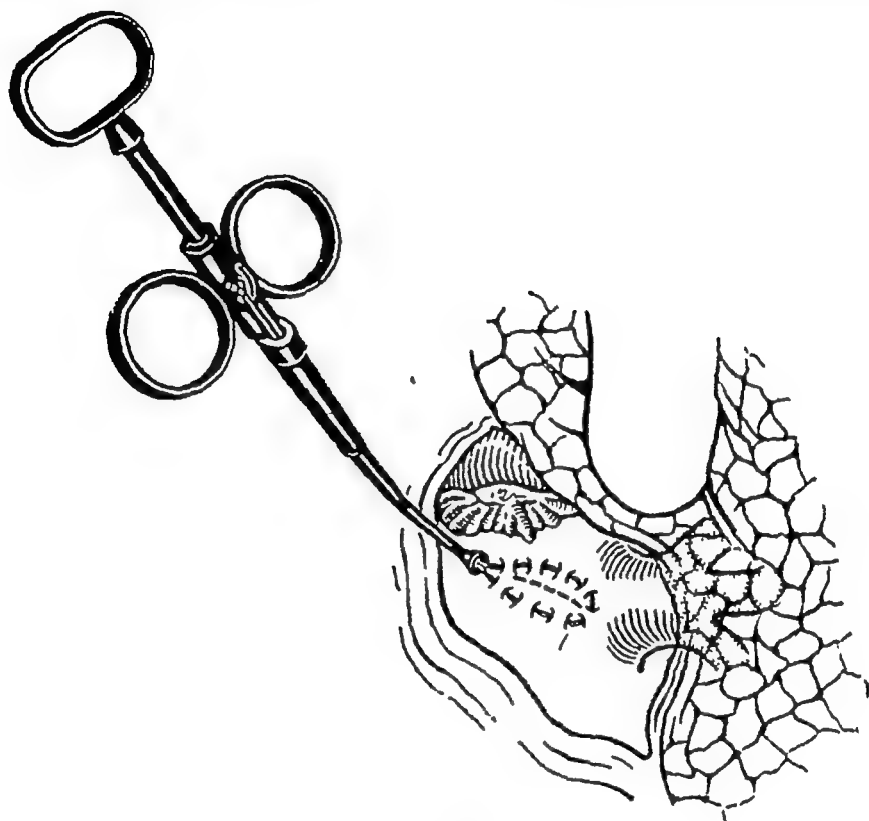


FIG 47

The use of a Rumel snare. As the appendage is vestigial an oval purse-string has been made on the wall of the atrium between the appendage and the superior pulmonary vein, and the ligature is controlled by gentle closure of the snare up which it is threaded. The dotted line shows the incision to be made in the myocardium.

gently tight so that no blood escapes round it and, as it is finally withdrawn, the purse-string is pulled tight and the closure reinforced with a few deep interrupted sutures.

Whether the heart is entered via the appendage, or directly as just described, no bleeding should occur that is not designed. If the appendage is used, as in the great majority of cases it is, the surgeon releases the clamp with his left hand as his finger passes into the

atrium. As it approaches the mitral valve some regurgitant jet is usually felt, and its nature and extent is carefully judged both before and after commissurotomy. The valve itself is palpated, and the degree of stenosis and pliability of the cusps estimated. After this has been done the finger tip is passed into the valve orifice and an attempt made to split first the posterior and then the anterior commissures as completely as possible. This proves possible most often in valves which are still diaphragmatic in shape, and there is much variation in the ease with which it can be done: the cusps sometimes parting effortlessly sometimes with an audible snap but usually requiring considerable force which must always be carefully controlled.

During separation of the anterior commissure the left hand is used for counter pressure on the front of the heart. Great care must be taken not to tear a cusp or to rupture one of the chordae or serious regurgitation will inevitably ensue. The finger must not remain too long in the valve orifice as it completely arrests the blood flow and the pressure drops momentarily to zero.

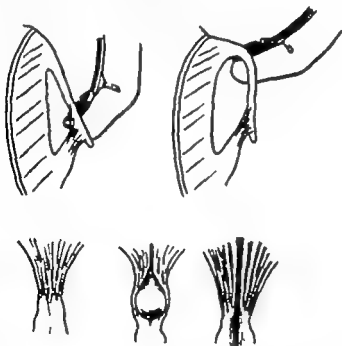


FIG 48

Above The tip of the valvulotome is engaged in the mitral orifice in the case of a much retracted cusp and the commissure cut upwards as it is lifted away from the myocardium.

Below The finger is passed down through the valve orifice to separate and free the matted chordae.

After splitting of the commissures, the finger is passed through the valve to separate the chordae tendineae if they are matted together and restore their mobility (Fig 48)

Although complete separation of the cusps may be accomplished with the finger alone, in very many cases this cannot be done, either the edge of the central orifice proving too tough to split, or separation occurring only so far out as the point of insertion of the thicker chordae, but not including the peripheral part of the commissure, i.e. that part which in retracted conical valves is closely in contact with the ventricular wall. In all such cases—and it is notable that their number increases with the experience of the surgeon—a valvu-

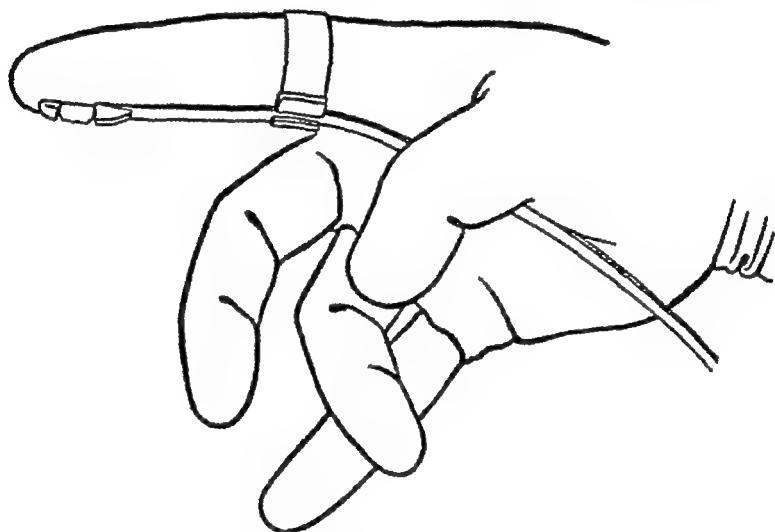


FIG 49

A Brock mitral valvulotome.

lotome must be employed. Many varieties exist, some in the form of guillotines, some as blades directly mounted on a ring; but the one preferred by the writer is that first devised by Brock, consisting of a flexible strip which is held to the index by a slotted finger ring so that it can be advanced or withdrawn at will to allow further palpation or digital splitting, and having at its end a small probe-pointed blade (Fig. 49). The probe is engaged in the orifice, and a little pressure on the blade then cuts the commissure. It can also be hooked under the deep edge of a much retracted valve and the portion lying parallel to the ventricular wall freed by drawing the knife-edge upwards (see Fig 48). The knife is not a substitute for the finger, or the finger for the knife, in most cases both are necessary,

supplement one another, and are used alternately until maximum separation and mobility of the cusps has been attained

At the end of the operation the valve orifice should be capable of accommodating two fingers. As the index is finally withdrawn from the heart the clamp is slipped back into place on the base of the appendage, but before this is done it is well to be sure no accidental tear has occurred which requires suture for it can more easily be controlled if the finger remains inside. The appendage is amputated, leaving only a flange of tissue beyond the clamp to be closed by a few interrupted mattress sutures and a final continuous stitch (Fig. 46c and d). The clamp is then removed and any leakage stopped by another stitch or two. The pericardial sac is thoroughly irrigated with warm saline until the fluid returns quite clear a dependent drainage hole is cut in it, and the rest closed with a continuous stitch. The lung is re-expanded, and the chest closed after a water sealed drain has been inserted.

Postoperative care

Before leaving the theatre the patient's limbs are examined to make sure no embolic arterial obstruction, which might require prompt surgical relief has occurred and as soon as consciousness returns it is reassuring to hear the patient speak a word or two move his arms and legs and to see that he has no abnormal ocular signs indicating a cerebral embolus.

The drainage tube may usually be withdrawn after 24 or 48 hours when an X-ray shows the lung to be fully aerated. Any subsequent pleural effusion is aspirated, but this is seldom necessary and atelectasis (should it occur) is treated by prompt bronchoscopy

The onset of atrial fibrillation is not uncommon postoperatively and is sometimes irreversible. In such cases, but not in those who were fibrillating before operation, the effect of quinidine should be tried after 10 days or a fortnight, and is often then successful in restoring normal rhythm. As the patient is already digitalized only a moderate increase in dose is usually required to keep control.

Pericardial effusions may cause a mild degree of cardiac tamponade with breathlessness, distention of neck veins and X ray evidence of an enlarged pericardial sac and if symptoms are severe it is very occasionally necessary to aspirate this.

Very similar symptoms are produced by the post valvotomy syndrome now well recognized, but whose cause remains obscure. As a rule this syndrome comes on about the end of the first week, or during the course of the second, but is sometimes longer delayed.

It is characterized by precordial discomfort, general malaise, a rise both of temperature and pulse rate, cervical venous distention and a pericardial friction rub. It is probable that the condition is post-traumatic in origin rather than due to reactivation of the rheumatic process, as has been suggested. The patient must be kept at rest until the symptoms and signs of venous distention subside. This they generally do spontaneously within a few days.

If no complications occur, and fibrillation if present is adequately controlled, patients can usually begin to get out of bed in the second week and start graduated activity, the extent of which depends largely upon the length of time they had to spend in bed before operation. Most will at once comment on their warmer hands and feet, on the removal of a sense of oppression and constriction across the lower chest, and on their ability to breathe more freely and to take deep satisfying inspirations. Improvement continues over many months and a true assessment of it cannot be made until a full six months have elapsed.

In 100 cases of mitral valvotomy observed for two years or more at the London Hospital results were classified as good in 57 per cent. These patients were able to earn their livings, do housework, and in some instances play games, in a fashion quite impossible before operation, and their whole range of activity was greatly extended. In 33 per cent. no substantial improvement followed, or early improvement was gradually lost over the subsequent two years. These were mostly patients with severely deformed and fibrotic valves to which function could not be restored by division of the commissures. Two patients were made worse by operation, because it resulted in increased regurgitation.

Seventy per cent. of patients were female, 30 per cent. male. Ages ranged from 17 to 55 years, but the average age was between 35 and 40 years. Fibrillation was present in 44 per cent. before operation, but in 62 per cent. afterwards. Twenty-nine per cent. had calcified cusps, and in half this number the calcification was heavy. Twenty per cent. suffered in some degree from the 'post-valvotomy syndrome' (*vide supra*). Operative mortality (i.e. death within three days) was 4 per cent., and another four patients died within three months of operation.

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